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Original Communications

MEDIASTINAL "TUBERCULOMA"

SURGICAL REMOVAL IN FOUR PATIENTS

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WITH the passage of years, detailed reviews of pathology and treatment have increased our common knowledge of mediastinal tumors. Mediastinal granulomas have received little attention, however, which undoubtedly bespeaks their rarity.

In earlier reports on mediastinal tumors, the differential diagnosis from mediastinal masses due to tuberculosis or syphilis is mentioned. The impression is gained that the distinction should not have been difficult since concomitant signs of specific disease would be present in addition to the central chest lesion.[1, 2] Apparently, Piazzi-Martinni[3] was the only author actually to describe cases of mediastinal tuberculoma and gumma. Heuer[4] has excluded both tuberculous and nontuberculous mediastinal lymphadenitis from the consideration of mediastinal tumors. It was not stated how the former diagnoses were made. More recently, Grow and co-workers[5] discussed eight instances of "tuberculous lymphadenopathy" among seventy-three patients who were explored because of mediastinal lesions. No further details were given.

Only two descriptions of the surgical extirpation of mediastinal tuberculomas have been found. Grace's case[6] apparently was the first to be recorded. The pathologic diagnosis was unsuspected prior to removal. The patient had a slightly positive tuberculin reaction. No special search was made for tubercle bacilli in the specimen. The patient was well at last report. Blades and Dugan[7]

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removed a posterior mediastinal tuberculoma, the nature of which was unsuspected prior to pathologic examination. A tuberculin skin test apparently was not done. Microscopically, rare acid-fast organisms were found in the specimen. This patient returned to full military duty.

Paulson[8] has removed two "tuberculomas" in army personnel. There were no symptoms; in both, the unidentifiable mass was discovered by routine roentgen study; in both, the tuberculin skin test was positive. At present, Paulson has a conservative attitude toward removal. He believes eventual calcification probable and fears trauma to surrounding structures because of extensive adherence, and possible spread of a tuberculous infection.

Blades[9] mentions two tuberculomas removed at operation in his report from Army chest centers. One case was his own; the other apparently was one of Paulson's.

The paucity of these reports leaves the questions of accurate preoperative diagnosis and of treatment largely unanswered. For these reasons four cases hitherto unreported are presented.

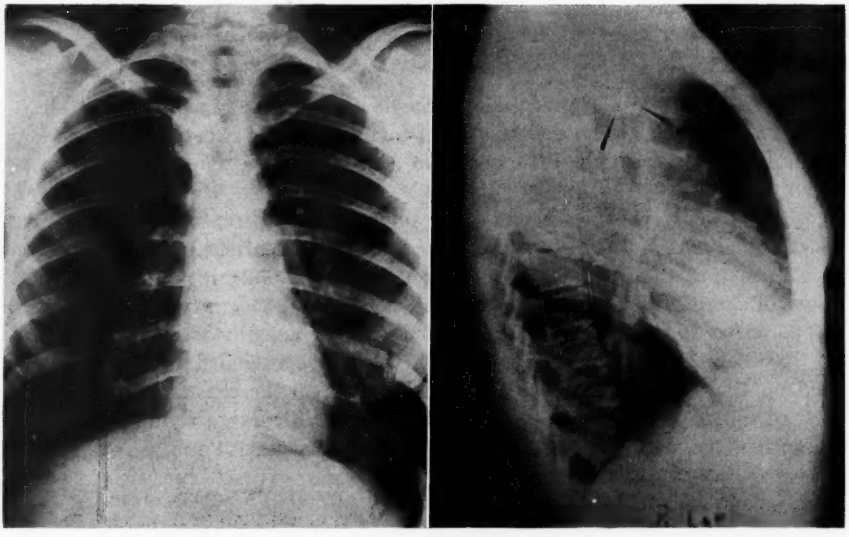

Fig. 1 (Case 1).—Roentgenogram, posteroanterior and right lateral projections. There is a globular mass, 3.5 cm. in diameter, in the right upper mediastinum. In the lateral view, it overlies the trachea. There is no evidence of tracheal compression.

CASE REPORTS

CASE 1.—V. M., a white, married woman, aged 27 years, was admitted to Letterman General Hospital in May, 1947. For approximately one year she had complained of a vague pressure in the chest and intermittent delay in the passage of food. There was slight progressive dyspnea on exertion. Six months prior to admission a general roentgen survey revealed a small mass in the right chest (Fig. 1). A teratoid tumor or a bronchiogenic cyst was suggested. Physical examination and laboratory studies were noncontributory. A tuberculin skin test was inadvertently omitted.

Right posterior thoracotomy was performed on May 19, 1947, through the fourth intercostal space. The upper lobe was adherent to a globular mass which measured 3.5 by 3 cm. The tumor was cystic and the wall was thick and yellowish in color. It was situated lateral to the trachea and was adherent to this structure and to the posterior aspect of the innominate artery. The vagus nerve coursed deep within the capsule and was sacrificed (Fig. 2). Medially and inferiorly the mass was bound down to several calcified lymph nodes which extended in a chain anterior to the trachea and toward the left. Due to extreme adherence, the nodes to the left of the trachea were not removed.

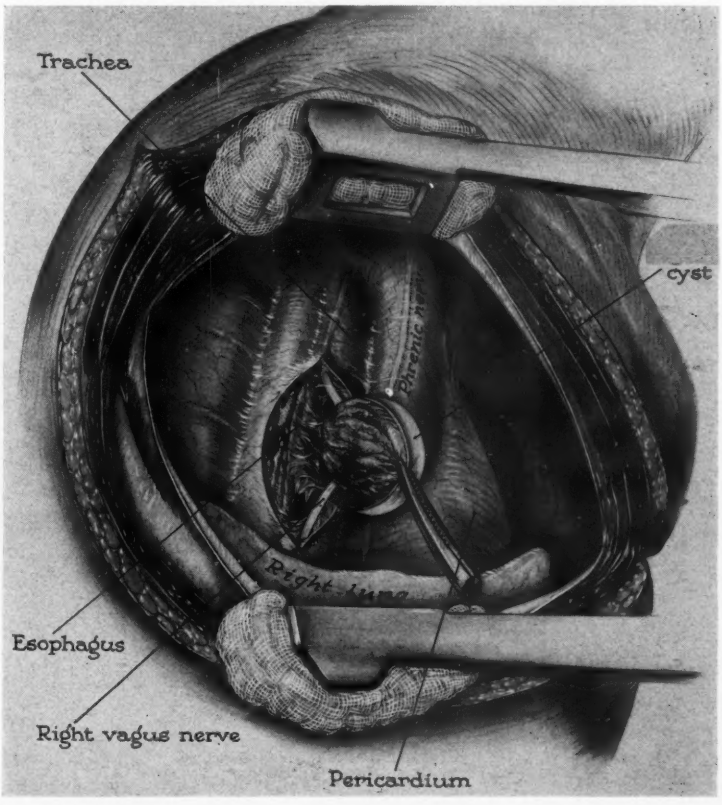

Fig. 2 (Case 1).—Drawing at operation showing position of mass in relation to mediastinal structures.

The postoperative recovery was uncomplicated and the patient left the hospital twelve days later.

Grossly, the cyst contained white caseous material. Numerous flecks of calcium were encountered in the contents and also in the thick fibrous wall. Microscopic examination showed fibrosis and chronic inflammation (Fig. 3). No epithelial lining could be seen. Culture of the contents of the cyst was negative for acid-fast organisms.

Case 2.—J. L., a white man, aged 19 years, had no symptoms referable to his chest. On roentgen examination prior to separation from the Army, a left-sided, oval, anterior mediastinal mass was discovered (Fig. 4).

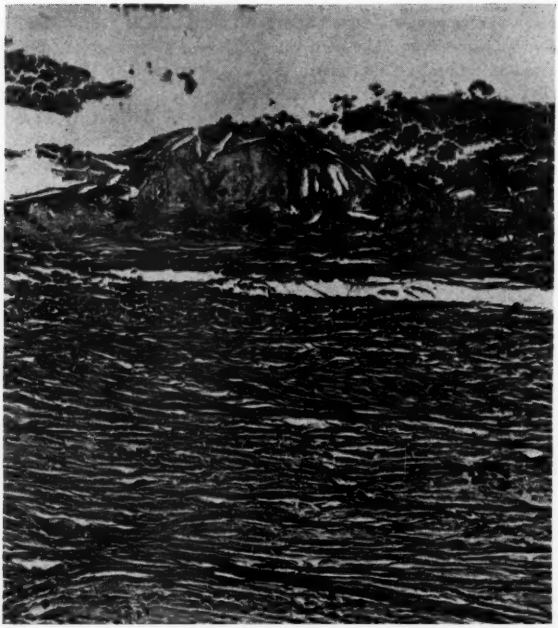

Fig. 3 (Case 1).—Photomicrograph ×100. Fibrous capsule showing cellular infiltrate and calcific deposits surrounding zone of caseation.

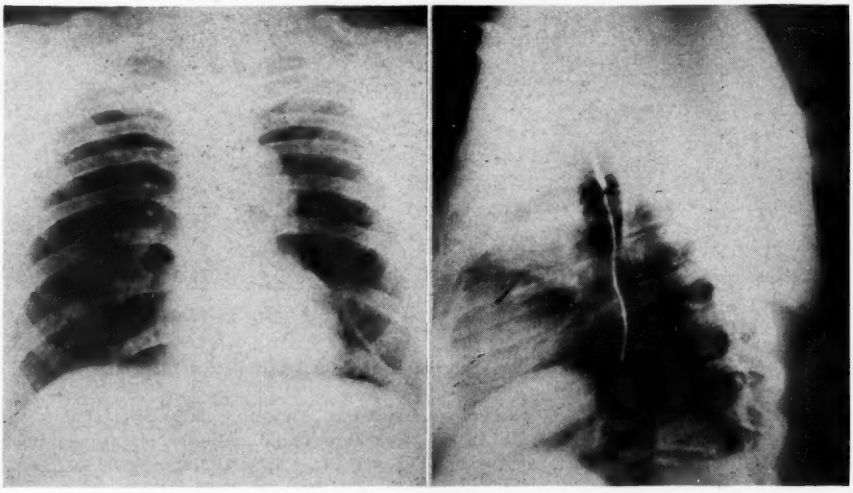

Fig. 4 (Case 2).—Roentgenogram, posteroanterior and left lateral projections. An oval mass measuring 2.5 by 5 by 5 cm. is present in the left anterior mediastium. There are small scattered areas of increased density suggestive of calcification.

Except for moderate obesity, the physical examination was unrevealing. There was no lymphadenopathy. The blood and urine were normal. The basal metabolism rate was −14. Both first and second strength PPD tuberculin tests were negative prior to and following operation. The coccidiodin skin test, 1 to 1000, was negative. The scanty mucoid sputum was inoculated into a guinea pig and no acid-fast bacilli were found.

Serial roentgenograms showed no change in the size of the tumor. Suggestive calcified densities were seen in some of the films and three possible diagnoses were entertained: dermoid cyst, tuberculous lymph nodes, and tumor of the lymphoma group.

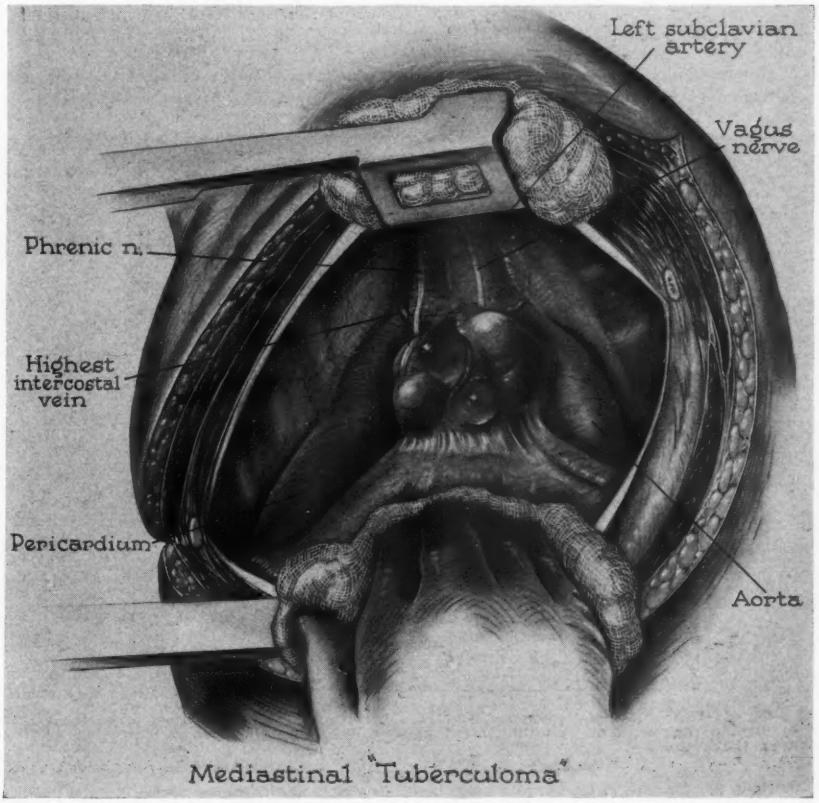

Fig. 5 (Case 2).—Drawing at operation showing lobulated and cystic mass in the left hilar region.

Left posterior thoracotomy was performed on June 13, 1947. There were no adhesions. The upper lobe was normal except for a small irregular subpleural calcified area in the anterior segment. The encapsulated mediastinal mass lay at the hilum beneath the mediastinal pleura and within the curve of the aortic arch. It measured 8 by 5 cm., was lobulated and partially cystic (Fig. 5). The vagus nerve was carefully dissected free from the posterior capsule. The phrenic nerve which coursed over the anterior surface of the mass was stretched somewhat during separation. Inferiorly and mesially the mass was adherent to the pulmonary artery.

The postoperative course was uneventful. On discharge, the chest was normal except for paresis of the left diaphragm. The patient has remained well.

Grossly, the mass was encapsulated and nodular, suggesting the fusion of at least three lymph nodes. Creamy white fluid and caseous material were contained in the cystic nodules. Small plaques of calcium were present in the wall. Microscopically, there was extensive caseation necrosis with an occasional rim of lymphoid tissue remaining at the periphery. Some of the caseous foci were enclosed only by a fibrous wall which was also undergoing caseation necrosis. Scattered calcifications were present in both caseous and granulomatous areas. Some caseous foci were enclosed by a zone of epithelioid cells through which were scattered many multinucleated giant cells varying from Langhan's to the foreign body type. In one area, small epithelioid tubercles were identified in the remaining lymphoid tissue (Fig. 6).

Special stains revealed no acid-fast organisms and cultures of material from the mass were negative for tubercle bacilli.

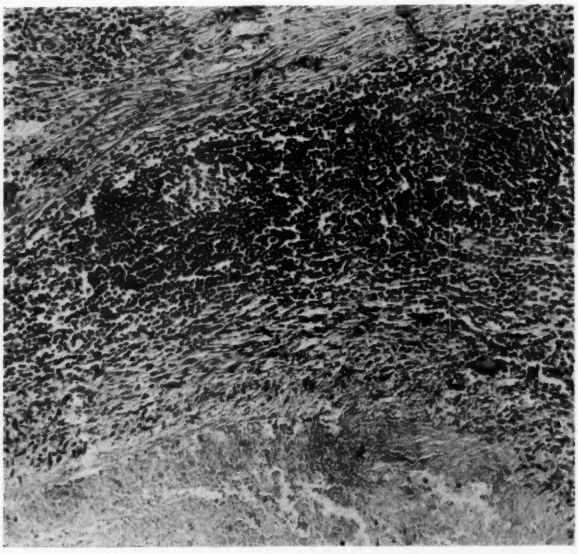

Fig. 6 (Case 2).—Photomicrograph ×100. A rim of lymphoid tissue is present. A zone of epithelioid cells with one multinucleated giant cell borders the area of caseation necrosis seen at the bottom.

CASE 3.—Private DiL., a white man, aged 22 years, was admitted to Letterman General Hospital on Oct. 1, 1946. He complained of an enlarging mass in the neck which had been present for one month. The patient had slight dyspnea when supine. Physical examination showed a soft, irregular, tender mass 5 by 2 cm. lying at the lower anterior edge of the left sternocleidomastoid muscle. The trachea was displaced slightly to the right. The mass moved but little on swallowing.

Exploration was done on the neck on October 23. The mass had penetrated the ribbon muscles and thyroid isthmus and was adherent to the trachea. It was dissected free and a connecting sinus was followed deep to the sternocleidomastoid muscle for a distance of 5 cm. This appeared to end blindly. The entire cavity was curetted, powdered with iodoform, and the skin and platysma closed. Examination of the specimen revealed a central abscess. Microscopically, the wall of the abscess showed epithelioid cells and

infiltration with polymorphonuclear leucocytes and eosinophiles. Rare giant cells of the foreign body type were present. Gram stains and acid-fast stains were nonrevealing. The guinea pig inoculation was negative for tuberculosis. The final pathologic report was encapsulated, nonspecific, chronic granuloma.

Continued swelling necessitated repeated aspirations. Three weeks later surgical drainage was performed, followed by daily irrigations. Three months following the original operation one irrigation caused severe cough. Lipiodol was then instilled in the sinus. There was continuity with a mass at the hilum of the left lung and a bronchial connection was demonstrated (Fig. 7). A secondarily infected bronchiogenic cyst was suggested. Earlier thoracic roentgenograms were then reviewed which had been previously reported as normal. The first of these had been taken elsewhere two months prior to development of the supraclavicular swelling. Each showed the presence of an ill-defined mass in the left hilum just below the aortic knob. The mass was enlarging slightly. Bronchograms produced six months after the original operation showed persistence of a Lipiodol puddle in the mass but no further bronchial connection.

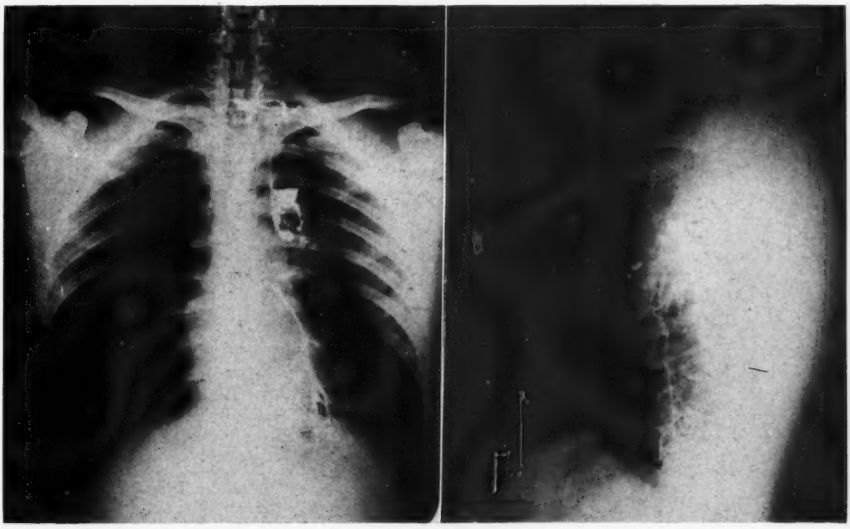

Fig. 7 (Case 3).—Roentgenogram in posteroanterior and left lateral projection following injection of Lipiodol into neck sinus. A narrow intrathoracic track is outlined leading to the mass in the left hilum where the Lipiodol is puddled. A generous bronchial communication is demonstrated.

The sinus in the neck eventually healed. Over a period of six months the patient's cough had increased appreciably.

On July 14, 1947, left-sided posterior thoracotomy was performed through the bed of the fifth rib. There were no adhesions. A 4 cm. nodular mass, partially hard and partially cystic, was found lying on the pulmonary artery incompletely covered by lung tissue. A firm cord was present beside the vagus nerve extending into the neck (Fig. 8). The vagus nerve and its recurrent branch were partially encased in the fibrous capsule of the mass. Both were dissected free with some difficulty. The phrenic nerve was inadvertently severed during separation. Removal of the entire mass was accomplished. Firm adherence resulted in a 7 mm. tear of the pulmonary arterial wall but this was repaired without difficulty. The tubular extension into the upper anterior mediastinum broke free in the region of the thyroid gland (Fig. 9). No lumen could be demonstrated nor could any connection with the bronchial tree be established.

The patient was discharged with an expanded lung. There was residual paresis of the left vocal cord and the diaphragm was elevated.

Gross examination showed that the specimen appeared to be a conglomerate mass of firm lymph nodes, some with caseous centers. Microscopically, the lesion showed granulomatous and inflammatory characteristics. Lymphoid tissue could be identified. Multiple caseous areas with occasional giant cells were widely separated by dense fibrous tissue. No acid-fast bacilli were recovered either on smear or culture (Fig. 10).

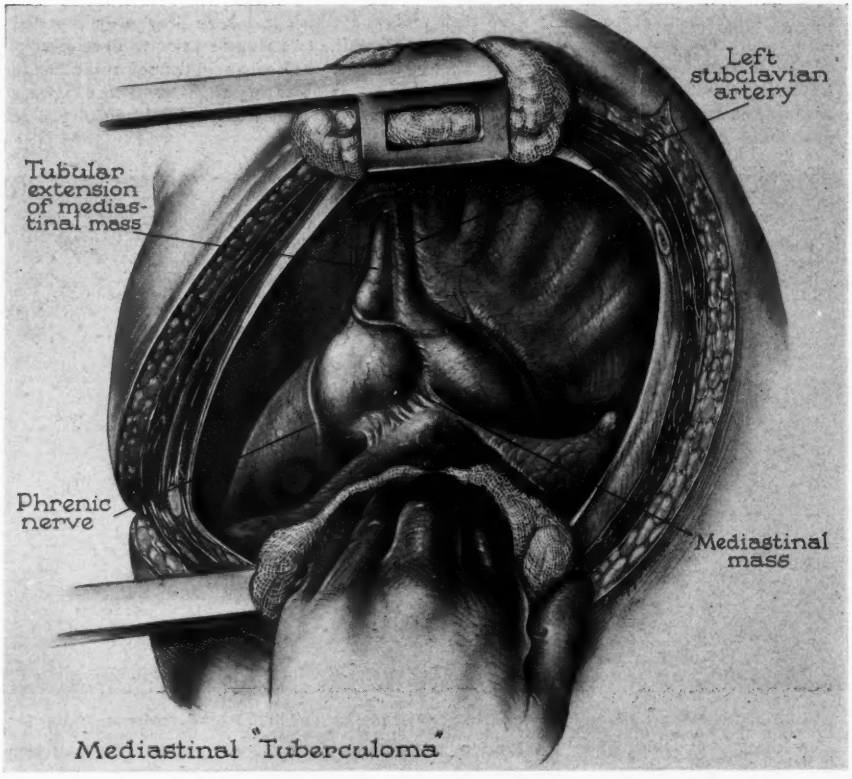

Fig. 8 (Case 3).—Drawing at operation. The nodular mass extends into the hilum. The tubular extension, crossed by the highest intercostal vein, disappears into the upper mediastinum.

CASE 4.—D. A., aged 34 years, was a white woman from Arkansas. She was first seen at the Stanford Medical Clinic in October, 1947, complaining of a cough of two years' duration. The cough was gradually increasing in severity and was paroxysmal and nonproductive. There were associated fatigue, poor appetite, a weight loss of six pounds, and increasing irritability. There were no night sweats, hemoptysis, or thoracic pain.

The patient had contracted syphilis at the age of 18, at which time she was treated for more than one year with "hip and arm shots." In March, 1947, both blood and spinal fluid Wassermann tests were positive and the gold sol curve was pathologic. Following penicillin therapy for one month the blood tests remained negative during the following year. The lumbar puncture was not repeated.

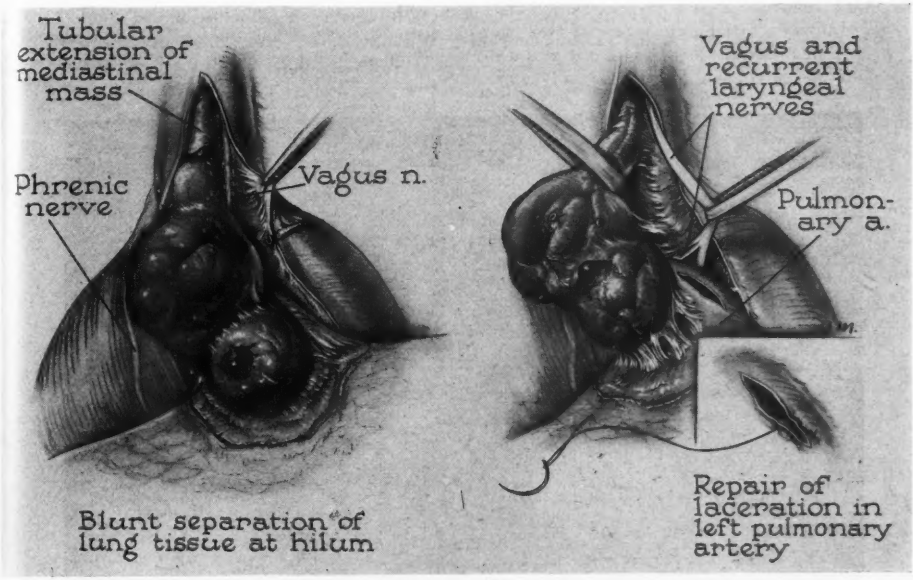
Tubular extension of mediastinal mass
Phrenic nerve
Vagus n.
Blunt separation of lung tissue at hilum
Vagus and recurrent laryngeal nerves
Pulmonary a.
Repair of laceration in left pulmonary artery

Fig. 9 (Case 3).—Details of dissection and of repair of lacerated pulmonary artery.

Fig. 10 (Case 3).—Photomicrograph ×50. Small tubercle undergoing central caseation necrosis in relic of lymphoid tissue.

Physical examination in October showed Argyll-Robertson pupils. There were no pathologic reflexes. A few clicking râles were heard over the trachea and the right upper lobe. There was a soft blowing murmur over the right second interspace and the second beat at the apex was reduplicated. The blood pressure was 120/80 in both arms. The pulse rate varied from 100 to 110 and the rhythm was regular.

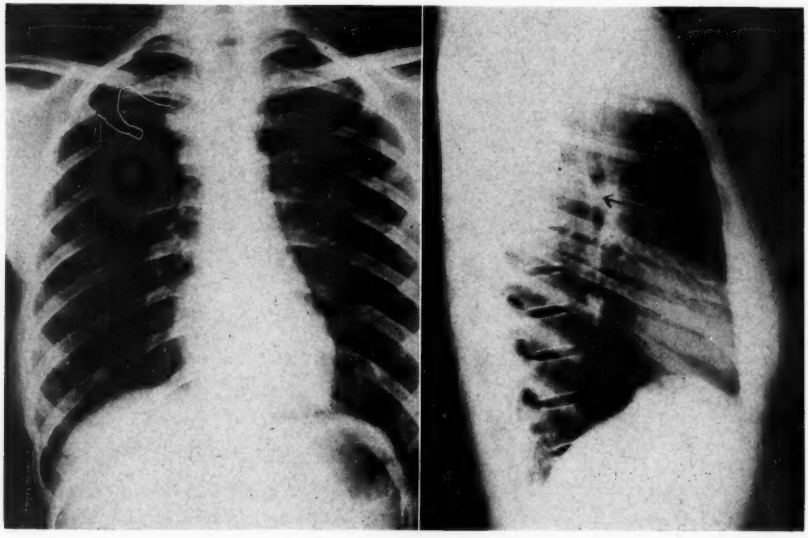

Fig. 11 (Case 4).—Roentgenogram, posteroanterior and right lateral projections. A smooth slightly lobulated mass is present in the right upper mediastinum. This has enlarged appreciably in three months time. In the lateral projection, the mass is immediately anterior to the trachea and there is suggestive compression of its lower portion.

Fig. 12 (Case 4).—Bronchoscopic view. The carina is pleated by external pressure. The tracheal lumen is reduced.

Laboratory studies revealed the following: hemoglobin, 15.5 Gm.; red blood cells, 3,920,000; white blood cells, 9,150; sedimentation rate, 52 mm. in one hour; urine, normal. The intradermal skin test to tuberculin, 1 to 1000, was negative; to coccidiodin, 1 to 1000, was negative, and to histoplasmin, 1 to 1000, strongly positive. The scanty sputum was re-

peatedly negative for acid-fast bacilli on concentrate examination. A roentgenogram of the chest showed a small, slightly rounded shadow in the mediastinum at the level of the right first interspace anteriorly. Fluroscopically it did not seem to pulsate and there was fairly satisfactory separation from the aortic arch. Laminagrams showed no encroachment on the lumen of the trachea. The patient refused further advice at this time.

During the ensuing four months, many of the patient's symptoms became aggravated. In particular, the cough became more constant, brassy, and only partially controlled by codeine. Posttussive dyspnea and wheezing developed, and faint oral stridor could be heard. Roentgenographically, the mediastinal mass had increased in size (Fig. 11). Bronchoscopy (P.C.S.) revealed extensive anterolateral pressure on the trachea and orifice of the right stem bronchus, the lumens being reduced approximately 30 and 50 per cent, respectively. The carina was accordion pleated (Fig. 12). The walls were smooth and the bronchoscope could be maneuvered past the obstruction, which pulsated slightly.

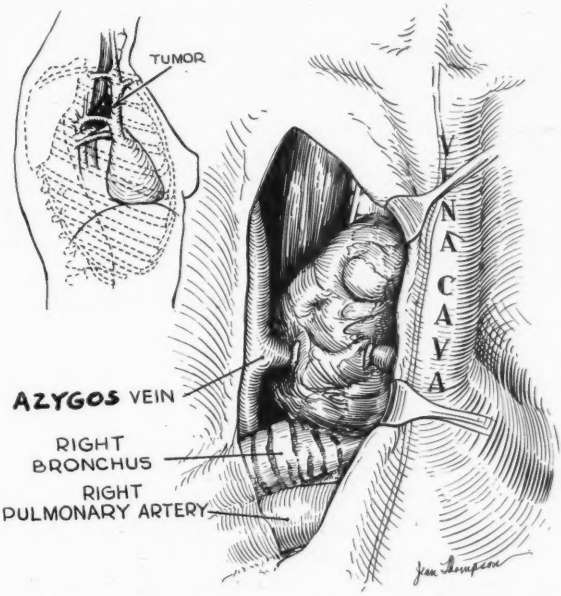

Fig. 13 (Case 4).—Drawing at operation. The mass is encroaching on the right lateral and anterior aspect of the lower trachea. The azygos vein has been divided for better exposure.

Exploration was recommended. The preoperative diagnosis was mediastinal tumor or, possibly, aneurysm.

Right anterior thoracotomy was performed* on March 3, 1948. A firm nodular mass 5 by 3 by 2 cm. was found (Fig. 13). The azygos vein was ligated to give better exposure. A portion of the mass was cystic and caseous material was extruded. Grossly, the mass was at least partially composed of matted lymph nodes. The portion which extended across the mediastinum to the left was not removed. A small tear in the superior vena cava was easily repaired. The pleural fluid removed on two occasions following operation was sterile and negative for tubercle bacilli on guinea pig inoculation.

Histologically, the tissue showed granulomas with central caseation. At the border of the necrotic tissue were numerous epithelioid cells, lymphocytes, plasma cells, and a few giant

*By Dr. Emile Holman.

cells. The granulomas were surrounded by hyalinized fibrous tissue with focal collections of lymphocytes. Small satellite tubercles were occasionally found. Pyogenic cultures were negative as were special stains to reveal acid-fast bacilli, spirochetes, and bacteria.

Eight months following operation the patient had practically no cough. Her appetite was poor and she continued to be nervous. The heart rate averaged 80 per minute. The thoracic roentgenogram was satisfactory. The intradermal tuberculin test, 1 to 100, was 2 plus.

PREOPERATIVE DIAGNOSIS AND INDICATIONS FOR EXCISION

Fairly complete information is now available on the eight known cases which have been subjected to surgery (Table I). Is it possible to make a more accurate diagnosis of mediastinal granuloma prior to operation? Survey of the available material suggests that it would be difficult. Concomitant evidence of active tuberculosis or syphilis might suggest the etiology of the mediastinal "tumor."[2] Roentgenograms certainly have not demonstrated "typical" masses; indeed, the shadows have uniformly mimicked more commonly encountered lesions. Evidence of scattered calcium deposits can be found in teratoid tumors as well as in granulomas. Undoubtedly, skin testing should be done more frequently. In the eight cases, a tuberculin skin test was applied in five and was positive in four. This percentage is appreciably higher than in the general population but does not necessarily indicate a conclusive diagnosis in the individual case. In patients from known endemic areas, coccidiodin and histoplasmin skin tests should be performed in addition to tuberculin.

In the final analysis, exploration will be necessary in nearly all cases, especially for the surgeon who follows the philosophy that unilateral mediastinal masses should be subjected to early surgical investigation.

Should an obvious granuloma be removed when discovered at exploration? Technical difficulties have been encountered because of inflammatory adherence to surrounding structures. Minor accidents such as tearing of vessels and sacrifice of nerves have occurred. On the other hand, five of eight patients had definite symptoms referable to the mass. In one, the symptoms were severe and there was partial tracheal obstruction. This case was one of four whose mass was in a similar location, the right superior mediastinum somewhat posteriorly. While two had no symptoms, there was no assurance that difficulties would not develop should the mass remain. Since the majority of patients had symptoms, we believe that granulomas should be removed if exploration is done. Even if the majority, or all such granulomas, are proved tuberculous in the future, experience to date is reassuring in that no active tuberculous infections have developed postoperatively.

PATHOLOGY

The lesions described previously were without question chronic granulomas. All presented a similar picture of fibrosis, round cell infiltration, epithelioid cell reaction, multinucleated giant cells, and varying degree of caseation necrosis. Seven of the eight cases showed residual lymphoid tissue which means that in so far as the mediastinum is concerned, the "tumors" have been composed of a granulomatous infection in coalesced lymph nodes. In this, mediastinal granulomas apparently differ from similar masses in the lung.

TABLE I

SURGEON	AGE (YR.)	SEX	SYMPTOMS	MEDIASTINAL LOCATION	TUBERCULIN	OTHER SKIN TESTS	PREOPERATIVE DIAGNOSIS
Grace	32	F	Dyspnea, pain	L. Ant. Sup.	Positive	Not recorded	Dermoid
Blades and Dugan	56	M	Dyspnea	R. Post. Inf.	Not recorded	Not recorded	Neurogenic tumor
Paulson, No. 1	33	M	None	R. Post. Sup.	Positive	Not recorded	Mediastinal tumor
Paulson, No. 2	21	M	None	R. Post. Sup.	Positive	Not recorded	Mediastinal tumor
Authors, No. 1	27	F	Vague pressure, dysphagia	R. Post. Sup.	Not done	Not done	Dermoid
Authors, No. 2	19	M	None	L. Ant. Sup.	Negative	Coccidiodin negative	Dermoid
Authors, No. 3	22	M	Dyspnea, cough	L. Ant. Sup.	Not done	Not done	Bronchiogenic cyst
Authors, No. 4	34	F	Severe cough, dyspnea	R. Post. Sup.	Preoperative 0 (1 to 1000) Postoperative + (1 to 100)	Coccidiodin –0 Histoplasmin ++++	Mediastinal tumor, (?) aneurysm

Whether or not the infection is due to the tubercle bacillus cannot be answered with such certainty. In seven of the eight cases, neither tissue stains, cultures, nor guinea pig inoculations revealed tubercle bacilli. In one case, a few acid-fast bacilli were found by special staining methods. Pulmonary granulomas due to coccidiomycosis have been reported,[5, 10, 11] presumably similar masses may occur in the mediastinum. Histoplasma capsulatum and syphilis may likewise produce caseating granulomas. As of the present moment sufficient clinical and laboratory investigations have not been done on these cases to prove conclusively the type of infection. When a granuloma is removed at operation, the tissue should be subjected to extensive cultural investigation and animal inoculation. Moersch and his co-workers[10] have stressed the importance of these steps in the diagnosis of obscure pulmonary lesions; they are no less valuable in determining the cause of a mediastinal granuloma. In essence, we do not believe that unquestioned proof of the tuberculous nature of these granulomas is at hand.

In regard to the absence of acid-fast organisms, one must admit the evidence of Griffith[12] and Feldman and Baggenstoss[13] whose investigations have suggested that viable virulent tubercle bacilli are rarely found in retrogressive, encapsulated lesions or in sclerotic lymph nodes.

SUMMARY

1. The available information on "tuberculomas" of the mediastinum has been summarized and four additional cases are reported in detail.

2. The possibilities of more accurate preoperative diagnosis are enumerated. As with other mediastinal masses, surgical exploration usually must be done in nearly all instances.

3. When a granuloma is diagnosed at the time of surgery, it is concluded that removal should be accomplished whenever possible.

4. The pathology has been discussed. The question is raised as to whether these granulomas are due to the tubercle bacillus or to other organisms. We believe that their tuberculous nature has not been unquestionably proved. Suggestions have been made as to future clinical and laboratory investigations which may aid in a more certain diagnosis.

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DISCUSSION

DR. DONALD L. PAULSON, Dallas.—My purpose in discussing Dr. Samson's paper is to present two cases that he recorded in his presentation. (Slides.)

This represents a mediastinal mass located posteriorly in the mediastinum. This was found in a routine survey and was asymptomatic. Operation was advised because it was a mediastinal tumor of undetermined type. It was found at operation to be densely adherent to the esophagus and the pericardium in the region of the atrium, and was removed with some difficulty because of this dense adherence. It was a cystic mass which was opened during removal and a thick, green, creamy material exuded from the mass. It was entirely removed, however, except for the calcified upper pole which was densely adherent to the mediastinal structures and it was considered wiser to leave this behind. This patient did well for three weeks postoperatively but at that time developed a pleural effusion on the right, with low-grade fever. The fluid was aspirated, smears and cultures for tubercle bacilli were made; examination of the contents of the cystic mass were also made for tubercle bacilli, and none were found in either the fluid or the contents of the mass.

This patient eventually recovered but had a period of about six months of low-grade fever, in my opinion due to a pleurisy.

The next slide reveals a mass in the upper mediastinum. Extensive studies were made in an attempt to make a definite diagnosis. Bronchoscopy revealed no evidence of a bronchiogenic tumor. Exploration was advised and carried out. What was interpreted at that time to be a mediastinal tuberculoma was found within the mediastinum densely adherent to the azygos vein, the superior vena cava, the right innominate vein, and trachea. The mass was removed with a good deal of difficulty because of the dense adherence to these structures, and a few openings were made in these large veins which were easily closed.

Pathologic examination of both of these masses revealed a chronic granulomatous type of lymphadenitis consistent with tuberculosis.

As a result of this experience it was my opinion that if a diagnosis of mediastinal tuberculoma could be made clinically, perhaps one would be wiser not to advise exploration because of the dangers of spread of infection, as occurred in the first case, or damage to the surrounding mediastinal structures to which these granulomas are usually densely adherent. At the present time we have two other patients under observation, one of which is asymptomatic, which we have followed for three years. It appears to be undergoing progressive calcification. The second patient is an older woman who I presume has a tuberculoma densely adherent to the esophagus which is producing symptoms of dysphagia. In the second case surgery probably will be advisable. In my opinion surgery is indicated in these mediastinal granulomas, tuberculomas, perhaps, only if they are symptomatic or if the diagnosis is in doubt.

DR. A. J. GRACE, London, Ontario.—I should like to say a few words about my case, the first one listed in Dr. Samson's table. This patient was operated upon in 1940 and the case was reported in considerable detail in the December, 1942, issue of THE JOURNAL OF THORACIC SURGERY. (Slides.) These slides show the sharply demarcated tuberculoma lying anterior to the left lung root and apparently causing some pressure effects here to account for her spasmodic dyspnea. It will be noted that this tumor is of considerable size, being rather larger, I believe, than any of those in Dr. Samson's cases. The slide illustrating the section of the tumor, following removal, gives some idea of its nature and structure.

It may be noted that this patient first had symptoms from her tuberculoma following a blow on the left chest. Following that she had pain in the region of the left breast, periodic dyspnea, and a low-grade fever at times. Serial x-ray examinations showed some definite variation in size of the mass.

It may be noted that in this case several medical consultants were opposed to operation because of functional considerations in the clinical assessment of the case. Everything possible, including deep therapy, was tried first before she was turned over to me for operative treatment. Early operation is certainly advisable in most cases of undiagnosable mediastinal tumors, and it remains the only method of establishing a diagnosis as well as of curing the condition in many of these cases.

I have been talking to this patient quite recently and find that she remains well and free from any noteworthy chest symptoms nearly nine years following the operation noted previously.

DR. WARRINER WOODRUFF, Saranac Lake, N. Y.—I should like to ask about the definition of tuberculoma in a problem of this kind. These are certainly not similar to those we see in lung substance. Those in the lung are to all intents and purposes undrained tuberculous abscesses and are filled with an inspissated caseous material from which tubercle bacilli are recovered in large numbers. In not a single instance of Dr. Samson's cases were tubercle bacilli found, either in the cyst or in the sputum. I wonder if these cases should not be recorded in the literature under some other classification than tuberculoma.

We have recently had a case of a large posterior mediastinal cyst which I think would fit this description—a man, 60 years of age, with an old calcified lesion which looked as if he might have had tuberculosis years ago. It projected on both sides of the spine. So far as attachment was concerned, apparently it came from the region of the bifurcation of the trachea, although there was no communication. Pathologic studies are not complete, but I think this may be another case which would fit into Dr. Samson's description; but we did not call it tuberculoma.

DR. ALFRED GOLDMAN, Los Angeles.—I wish briefly to report an experience with a case of chronic granulomatous mass such as Dr. Samson showed, which had in it calcifications, and a sinus tract leading from the calcified area to the esophagus. This patient was sent to me as a possible carcinoma of the esophagus. Examination revealed a puckered red granulomatous mass involving one-half the circumference of the wall, and after going beyond the area one saw a few drops of pus from the opening in the esophagus. Biopsy showed chronic infection, and no acid fast bacilli were found. At operation glands were seen at the bifurcation of the trachea which contained about 15 c.c. of pus. The pus was sterile. Histologic examination of the mass was reported as nonspecific granuloma. The coccidiodin test was negative although the patient came from that area. The tuberculin test was not positive.

DR. SAMSON (Closing).—I agree with Dr. Paulson that if a diagnosis can be made many lesions give symptoms which are not diagnosed except by operation, and that course seems to be the best.

Dr. Woodruff did not see the paper, in which the word "tuberculoma" was in quotation marks. Personally I am very much undecided that they are tuberculomas, and as a matter of fact I suspect that many are not. Certainly they differ from the so-called tuberculoma of the lung.

Dr. Goldman's case is interesting. A granuloma usually is a specific entity if I remember correctly. While the etiology cannot always be determined I question whether we should use the term "nonspecific granuloma" in this generic sense.

ACCIDENTAL CYST OF THE CHEST WALL LINED WITH BRONCHIAL EPITHELIUM

Report of a Case

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INTRODUCTION

THE FOLLOWING case is presented as an unusual occurrence, which illustrates the reaction of bronchial tissue to injury. No identical case was noted in a review of the literature.

CLINICAL HISTORY

V. C., a 32-year-old white man, entered Halloran Veterans Administration Hospital on May 5, 1947, complaining of a tender mass in the right side of the chest wall which first appeared in April, 1945, and increased in size until his admission. Past history revealed that he was struck by antiaircraft shell fragments while on a mission over Germany in August, 1943. His plane was shot down, and the patient received wounds of the right side of the chest. Following capture he was found to have a right pneumothorax, and surgical removal of metallic fragments was done through a short posterior chest incision at a German prisoner-of-war camp hospital. According to the patient, he developed an "acute pleurisy" in the right side of the chest postoperatively and was hospitalized for a period of six months. The wounds healed, and thereafter he was asymptomatic except for occasional pains in the right side of the chest. On April 25, 1945, at the time of his liberation, he first observed a small swelling on the right lateral side of the chest wall, which was slightly tender. In the spring of 1946 he observed an increase in size of the mass and complained of increasing tenderness over this area. He also complained of slight exertional dyspnea which continued until the date of admission. There was no accompanying cough or hemoptysis. Physical examination was negative except for a stellate scar 5 cm. in diameter 2 cm. to the right of the ninth dorsal vertebra, posteriorly. Present in the right side of the chest wall in the posterior axillary line was a lemon-sized mass which was hard, slightly tender, and firmly attached to the chest wall over the eighth to tenth ribs. It was not attached to the overlying skin. X-ray examination revealed the mass to contain a metallic foreign body within it (Fig. 1).

The mass was removed on May 13, 1947, under local field anesthesia. At operation the bands of the serratus anterior and latissimus dorsi muscles were found immediately overlying the mass and firmly attached to its thick, gray-white capsule, forming a portion of the outer wall of the cyst. The muscle bands were split and separated by sharp dissection to reveal a spherical mass beneath, which was tense and slightly fluctuant. The posterior aspect of the mass was separated with difficulty and was adherent to the underlying ribs and intercostal muscles by a dense avascular tissue which radiated in a fanlike fashion, fusing with the muscle fascia and periosteum. This was dissected and the mass removed, leaving the floor of the defect formed by the dense scar tissue previously described.

It was evident at the time of operation that the sequel to injury had been a marked tissue reaction to the presence of a foreign body. This involved not only the structures in the rib cage, but also the overlying muscles and fascia.

Gross Appearance.—The sac was tensely distended and measured 4.5 cm. in diameter. Its outer surface was pink in some aspects, gray in others (Fig. 2). When the sac was opened,

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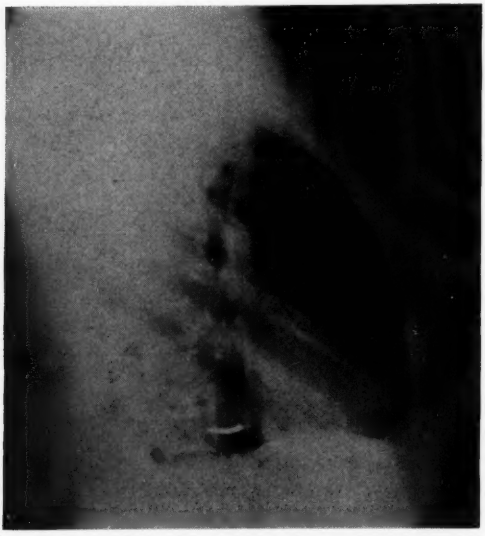

Fig. 1.—Roentgenogram, lateral view, showing metallic foreign body in superficial aspects of chest wall.

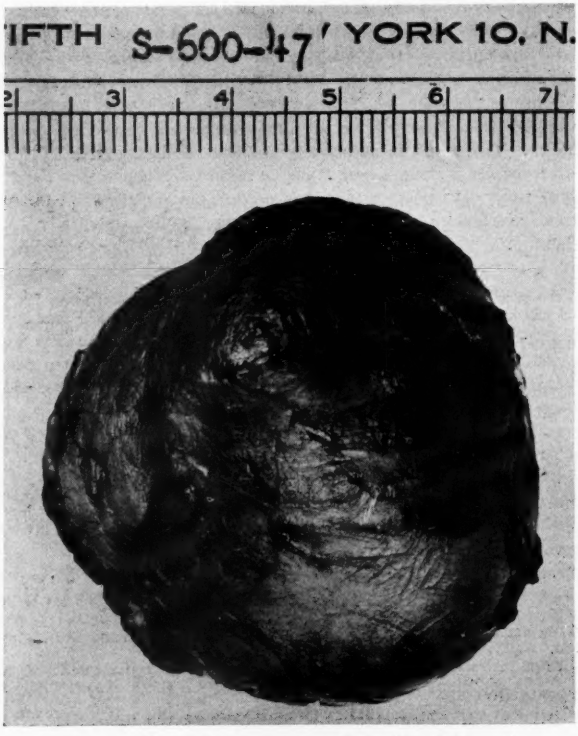

Fig. 2.—External appearance of foreign body cyst.

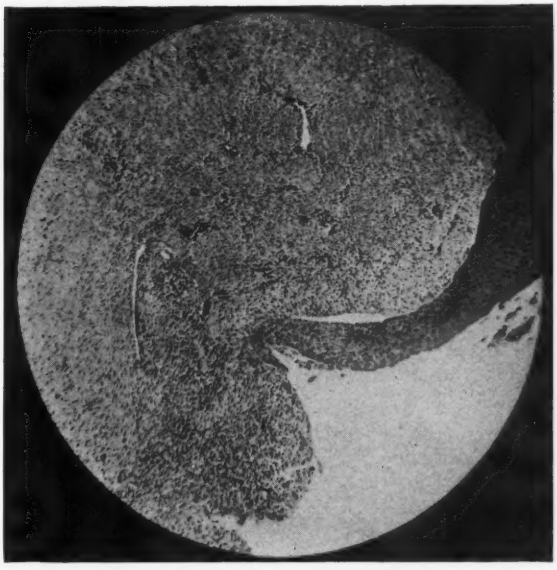

Fig. 3.—Photomicrograph of cyst lining showing stratified epithelium bordering vascular granulation tissue (×100).

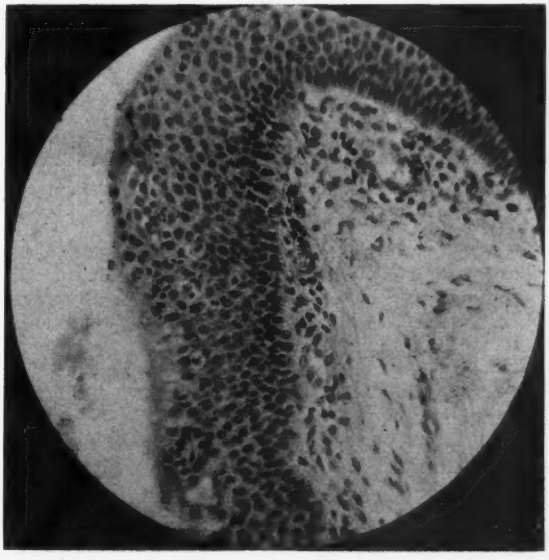

Fig. 4.—Photomicrograph of cyst lining near its transition from stratified squamous to ciliated columnar epithelium (×400).

it was found to contain approximately 15 c.c. of thick, tenacious, ginger-brown fluid. Contained within it also was a jagged sliver of steel shrapnel which measured 2 cm. by 6 mm. by 3 mm. It lay free within the lumen of the cyst. The wall of the sac measured 2 mm. in thickness, and its inner surface had a granular, red-brown appearance in some portions, and was lined by a smooth, gray membrane in others.

Microscopic Appearance.—Portions of the cyst wall were lined by stratified squamous epithelium (Fig. 3); other aspects were lined by a pseudostratified, ciliated columnar epithelium. Many of the cells in the latter portions had a goblet appearance (Figs. 4 and 5). Beneath the epithelium were dilated capillaries, histiocytes, plasma cells, and lymphocytes. In still other aspects of the wall the continuity of the stratified squamous epithelium was interrupted, and here the cyst wall was lined by a vascular granulation tissue composed of dilated capillaries, fibroblasts, collagenous fibers, many polymorphonuclear leucocytes, plasma cells,

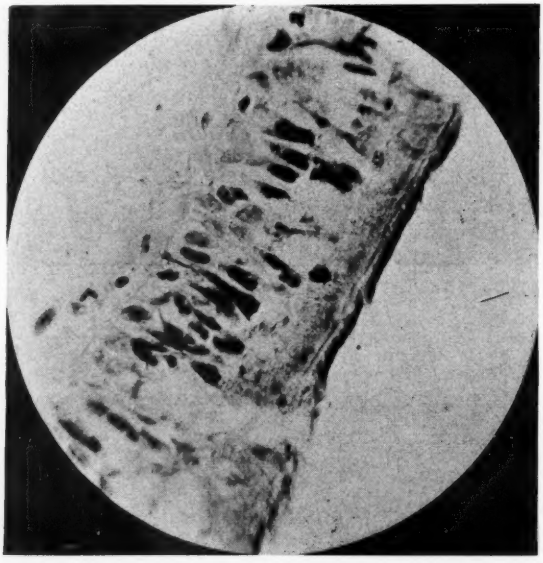

Fig. 5.—Photomicrograph of the mature ciliated columnar epithelium which lined portions of the cyst (×950).

occasional eosinophiles, and numerous macrophages heavily ladened with brown pigment. This granulation tissue extended for a short distance beneath the adjacent stratified squamous epithelium. The outer portions of the cyst wall were composed of dense collagenous connective tissue which extended between and separated the muscle bundles adjacent to the outermost portions of the sac.

Subsequent sections from other portions of the circumference of the cyst wall revealed changes similar to those already described.

DISCUSSION

The manner in which the cyst came to be lined by a mature bronchial epithelium is of great interest. Two possibilities present themselves:

1. The cyst may represent a remnant of a bronchopleural-subcutaneous fistula. That such a fistula might have occurred is indicated by the presence of air in the affected pleural space after the original injury. As the tract healed

the lumen may have become narrowed by the developing scar tissue in its wall, so that eventually it was completely obliterated. Unfortunately the early history of the injury is not reliable, and whether or not it was followed by a frankly suppurative empyema cannot be ascertained. Such a sequence of events, however, has been reported in analogous situations. Auerbach and Green[1] have observed the epithelization of healed and healing tuberculous cavities. In such cases the new epithelium is either columnar or stratified squamous in type. These authors also quoted Pagel and Koch as having demonstrated the epithelization of pulmonary-cutaneous fistulas in cases of tuberculosis. Lisa and Rosenblatt[2] emphasized the re-epithelization of bronchi by ciliated columnar epithelium in cases of bronchiectasis. In all these situations one source of the new epithelial lining is from the healthy bronchial epithelium bordering the diseased areas. In the case of tuberculous cavities it is derived from the draining bronchi. In bronchiectasis small islands of intact epithelium may serve as an additional source of lining cells.

At the operation no such sinus tract was demonstrated, but the dense scar tissue which bound the cyst to the outer layers of the periosteum and intercostal muscles may represent the healed remnant of a fistulous tract.

2. The bronchial epithelium may have been carried to its definitive site by the jagged projectile. To accept this possibility it must be assumed that the journey was of such a nature that it did not destroy all of the transported tissue. Since only small numbers of healthy pavement cells are necessary to effect re-epithelization, this mechanism is a distinct possibility.[2, 4] The progress of the fragment through the chest was obviously a slow one. In contrast to rifle bullets, shell fragments are quickly spent. As the shell fragment collided with the ninth rib its progress was materially impeded. That it came to rest within the body also emphasizes its slow rate of travel.

Upon its arrival on the new site in the superficial aspects of the chest wall the epithelium may have come to line the existing tract or the space formed by the hematoma surrounding the foreign body.

It is well known that bronchial epithelium, when it has been subject to the insults of disease or injury, often becomes stratified squamous in type. Two factors served to make the new site a similarly unfavorable environment. The first of these was the foreign body reaction in the cyst wall. In addition, the active secretion of mucus by the epithelium considerably increased the pressure within the cyst. The presence of the stratified squamous epithelium in portions of the lining membrane represents, then, an expected reaction to injury. Miller[3] quoted Baraban as believing the transition from ciliated columnar epithelium to stratified squamous epithelium to be due to low-grade inflammation which leads to loss of cilia and the appearance of mucus in the cells. Both Miller and Baraban agreed that in the lung coughing probably plays a prominent role in this transition. Condon[4] believed that the stratified squamous epithelium which appears after injury to bronchial epithelium, as in mechanical trauma, chemical irritation, or disease is a pseudostratified form of normal respiratory epithelium. It represents not a metaplasia, but a protoplasia and is an early regenerative

form of epithelium. He stated further that the rapidity with which it is converted to ciliated columnar epithelium is dependent upon the state of the subepithelial tissue. In the presence of inflammation or irritation the conversion is a slow one. If Condon's hypothesis is a correct one, only a small amount of basement cells need have been the source of the epithelium which lined the cyst which we are reporting. From them the stratified squamous epithelium was developed, the transition to columnar epithelium representing the final step toward maturity.

That portions of the lining epithelium in our case were physiologically as well as anatomically mature is evidenced by the recent enlargement of the cyst during the year prior to its excision. The contents of the cyst were viscid, tenacious, and had a mucoid consistency. The ability of the epithelium to take root upon a damaged surface and to continue its specific function in a new location speaks for its resilient vitality.

SUMMARY

1. A foreign body cyst in the superficial tissues of the chest wall has been observed. It was lined by pseudostratified ciliated columnar epithelium, stratified squamous epithelium, and granulation tissue.

2. The cyst may have arisen as an accidental tissue transplant or may represent the remnant of a sealed-off bronchopleural subcutaneous fistula.

3. The reaction of the aberrant bronchial epithelium to injury mimics similar reactions in the diseased or injured lung.

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ACTINOMYCES IN PUTRID EMPYEMA

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THE manner of development of pulmonary actinomycosis and our ideas concerning the pathogenicity of actinomyces for man require clarification.

Pulmonary actinomycosis may occur in three ways: (1) by direct extension from the abdomen through the diaphragm, (2) as a metastatic infection, (3) by inhalation.

The occurrence of infection of the lung through the diaphragm from an actinomycotic abscess of the liver perforating directly into the lung has been observed; this mode of infection of the lung has been well established but is not common. It is also well known that actinomyces may spread through the blood stream to produce hematogenous lesions in the spleen, kidneys, and bones, and it is possible for the lungs to become involved in the same way. However, in most cases of pulmonary actinomycosis, particularly in the earlier stages, the disease is found confined to the lungs, pleura, and chest wall, indicating that most cases of pulmonary actinomycosis have their origin in the airway, that is, that the infection is bronchogenous.

Actinomyces may conceivably be implanted in the lung by direct inhalation of the organisms from the air into the pulmonary alveoli, or they may be inhaled together with detritus or exudate from the mouth or throat in association with other organisms. This material may then become impacted in one of the smaller bronchi to produce infection of the lung. It is a peculiar fact that actinomyces, pathogenic for man, have never been found free in nature.[5, 7] This, together with the apparent low pathogenicity of the organism, which will be discussed later, and the absence of evidence that the disease is contagious,[7] make it extremely unlikely that infection of the lungs takes place by the direct inhalation of actinomyces floating free in the air.

A site of predilection for actinomycosis is the jaw; the infection usually arises in the gingiva, which is the common nidus for the actinomyces. Here the actinomyces are found together with other mouth organisms, many of which also are anaerobes. It has been a common observation, moreover, that actinomyces occur with great frequency in this location in persons either with no visible disease of the gums or with what appears to be simple chronic gingivitis or periodontoclasia (pyorrhea) without any evidence of actinomycosis as such. In addition, actinomyces have been found in the plugs within tonsillar crypts without evidence of actinomycosis of the tonsils.[1-5]

In spite of the fact that the actinomyces isolated from such cases were anaerobic and adhered strictly to the cultural characteristics of the pathogenic form described by Israel and Wolff[6] and known variously as *Actinomyces bovis*, *A. hominis*, and *A. israeli*, one can hardly consider them to be acting as pathogens

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in these cases. The difficulty that has been experienced in producing lesions in animals by the injection of these supposedly virulent organisms also indicates their low degree of infectiveness. It appears most probable that given an organism of such low-grade virulence, infection of the lung through the air passages must occur through the impaction in the smaller bronchi of particulate matter from the mouth or throat containing these organisms. If this material is not expectorated over a period of time, the likelihood of infection of the contiguous bronchus and the segment of lung that it supplies becomes apparent.

Under such circumstances it is rather to be expected that organisms carried down with the exudate or detritus from the mouth, together with the actinomyces, play a role in the ensuing infection. It has generally been assumed that bacteria found in the lungs in association with pulmonary actinomycosis are secondary invaders. But if the course of events postulated previously is accepted as the true pathogenesis of the disease, it must follow that these supposedly secondary invaders were present from the beginning, together with the actinomyces. Because of the difficulty which has attended the production of pulmonary actinomycosis by the intratracheal injection of pure cultures of actinomyces,[5] it seems quite probable that these other nonspecific organisms play a part in the production of the infection by symbiosis.

A very similar course of events takes place in the production of putrid abscess of the lung. It is well known that the organisms responsible for this disease are not very virulent by themselves and that they are common inhabitants of the mouth or throat, where they may produce little or no disease. It has been amply demonstrated that this disease comes about from the inhalation of particulate material containing the anaerobes from the mouth or throat into the bronchi and that prolonged contact of the inhaled material with the contiguous bronchial mucous membranes leads to infection of the bronchus and the segment of lung which it supplies, to produce the localized area of gangrenous bronchopneumonia that leads to the putrid lung abscess.

A similar difficulty has been experienced in the attempts to produce putrid lung abscess by the injection of any one of the anaerobic organisms into the bronchial tree, and it has been generally concluded that in this disease symbiosis of the organisms is probably required for its production.[9] No one will consider that the mixture of organisms found in acute putrid lung abscess represents a host of secondary invaders. They are all part of the original infection even though a good many of the organisms are comparatively avirulent and by themselves would not be responsible for infection.

A parallel between the pathogenesis of pulmonary actinomycosis and putrid lung abscess is found in the three cases reported in this paper. In each of them, there was a putrid empyema secondary to the rupture of a putrid lung abscess. These contained the usual flora that are encountered in lung abscess. In addition there was found in the pleural exudate of each of them the anaerobic *A. israeli (A. bovis)*. In one case the pulmonary infection followed tooth extraction, and in the other two there was advanced periodontoclasia with purulent gingivitis. In one of these the actinomyces were searched for and found in the exudate about the gums.

CASE REPORTS

CASE 1 (M. S. H. No. 431467).—W. W., a 46-year-old lithographer, was admitted complaining of severe right-sided chest pain of four weeks' duration. His past history was not remarkable. Five months before admission he had had several molar teeth extracted under local anesthesia. His teeth were considered bad, he had had pyorrhea about them, and one or more teeth were broken in the process of extraction. Three months before admission the patient began to feel ill and developed a cough, which at first was nonproductive, but later he coughed up a small amount of dark blood. During the month preceding admission to the hospital he developed severe, sharp pains in the right lower part of the chest and exertional dyspnea. He had occasional elevation of temperature, up to 100° F. on four occasions and experienced night sweats. An unsuccessful attempt to aspirate the right side of the chest had been made three days before admission.

Examination disclosed a well-developed and well-nourished, pale, white man in no acute respiratory distress. Temperature was 100.8° F., respirations were 24 per minute, and pulse rate was 96 per minute. He wore an upper partial denture, and he had moderate pyorrhea alveolaris. Several teeth were carious. His breath was not foul. The fingers showed slight clubbing. There was flatness over the posterior part of the right side of the chest up to the spine of the scapula; the breath sounds were suppressed over this area.

The hemoglobin was 82 per cent, the white blood cells numbered 12,000, and the differential count was normal. X-ray examination of the chest showed a small collection of fluid loculated over the posterior portion of the right upper lobe with marked thickening of the pleura over the entire right lung. Bronchoscopy was negative except for the presence of a small amount of thin, nonfoul secretion from the orifice of the right upper lobe bronchus. This did not recur after suction. Bronchography with Lipiodol revealed the branches of the lower lobe to be crowded together by the loculated fluid. Aspiration in the third intercostal space, paravertebrally, yielded a small amount of foul pus. A thoracotomy was performed, and a well-localized putrid empyema was found adjacent to the spine. The empyema space was shallow, and the visceral and parietal pleura were greatly thickened. In the mid-portion of the exposed visceral pleura there was an area of about one-half inch in diameter which was soft and presented a superficial erosion.

A smear of the putrid material obtained at operation revealed gram-negative bacilli, streptobacilli, and spirilla. Only *A. israeli* was grown from the empyema fluid.

The wound healed without incident. During two years of observation there was no recurrence of the symptoms, and the wound remained well healed. A film of the chest as early as one month after the operation showed no residual shadows.

CASE 2 (M. S. H. No. 444882).—L. C., a 35-year-old writer, was admitted with the chief complaint of pain in the right side of the chest of two weeks' duration and foul sputum for one week. His past history was noncontributory. His present illness began rather abruptly two weeks previously with severe pleuritic pain in the right posterior part of the chest. This was treated at another institution as a bronchopneumonia with sulfapyridine. One week after the onset, fever and foul sputum were noted, and he was then transferred to the Mount Sinai Hospital.

He was a well-developed and well-nourished, feverish young man in no acute respiratory distress. Temperature was 101.2° F., pulse rate was 92 per minute, and the respirations were 24 per minute. The dental consultant reported poor oral hygiene, calcareous deposits about the teeth, and periodontoclasia. His breath was not foul, and there was no clubbing of the fingers. There was dullness and diminution of breath sounds and fremitus in the right posterior part of the chest below the angle of the scapula. The entire right side of the chest was tender.

X-ray examination of the chest revealed a fusiform shadow in the region of the short fissure. Bronchoscopy revealed a small amount of thin pus with a peculiar sweetish odor issuing from the mesial and apical branches of the right lower lobe bronchus. Cultures of the sputum revealed hemolytic streptococci. During the following week he had a low-grade fever and the sputum had no odor. Repeated chest aspirations were negative. Finally,

aspiration in the sixth interspace just medial to the posterior border of the scapula yielded a small amount of thin, faintly odorous pus, and a thoracotomy was performed by resecting the eighth rib. The lung was covered with shaggy exudate containing a number of small collections of thin, turbid, foul pus. No pulmonary lesion was found.

The immediate convalescence was uneventful, the sputum disappeared, and the temperature promptly returned to normal. Bronchoscopy one week later was negative, and Lipiodol injected into the wound failed to enter the bronchial tree. Microscopic examination of the empyema wall revealed only acute and chronic inflammation. A culture was not made.

The patient was re-admitted five months later. He stated that the wound had continued to drain for two months and then closed. He had felt well until four days before admission, when he developed fever and pain in the scar. Physical examination revealed a fluctuant swelling in the center of the thoracotomy scar. This was incised and a cavity extending into the pleura, measuring about five by three inches, was found. No fistula could be discovered.

Microscopic section of the empyema wall revealed acute and chronic inflammation, with giant-cell formation. The wound healed shortly after discharge from the hospital, and the patient felt well for the following eight months. He then experienced chest pain and began to expectorate a small amount of yellowish, nonfoul sputum, which on culture, showed enterococci, *Streptococcus viridans* and *Staphylococcus albus B.*

Physical examination showed slight clubbing of the fingers. Once again the teeth were in poor condition. The scar was bulging and fluctuant. Incision of the swelling yielded a few ounces of foul pus. The cavity was smooth walled, and the operator was not certain whether it was intra- or extrapleural. No fistula could be demonstrated. Culture of the pus revealed *A. israeli.*

The patient returned to the hospital for further study one month later. Repeated examinations of the sputum, of the thin, mucoid secretion of the sinus tract, and smears and culture of material obtained by bronchoscopy revealed no actinomyces. He was discharged with the wound healed. When last heard of through the follow-up clinic, three and one-half years later, the patient was well, had had no recurrence of symptoms, and the wound was well healed.

CASE 3 (M. S. H. No. 562568).—M. P., a 55-year-old white man, laborer, was admitted complaining of constant pain in the left side of the chest for three months. He had been hospitalized elsewhere eight months previously for cough and pain in the left side of the chest of one month's duration, and after one month of observation, was discharged. Three months prior to admission he noted severe pain on the left anterior and posterior side of the chest, shortness of breath, and a cough productive of small amounts of nonfoul whitish sputum. He had had profuse night sweats for two weeks.

He was a well-developed, well-nourished, dyspneic man, coughing up greenish, nonfoul sputum. Temperature was 103° F., the pulse rate was 120, and the respirations 24 per minute. Many teeth were missing, many of the remainder were carious, and there was a severe purulent gingivitis. The trachea was deviated to the right. There was dullness to flatness over the entire left lower lobe where breath sounds were absent, and there were diffuse wheezes throughout both lungs. The fingers were clubbed.

The hemoglobin was 56 per cent, and the white blood count numbered 17,100 per centimeter with 89 per cent polymorphonuclear leucocytes. Culture of the sputum yielded *Str. viridans, Str. hemolyticus* and *Micrococcus catarrhalis.* A chest film revealed an effusion in the left lower part of the chest, loculated anteriorly and lateral to the heart. There was an additional loculation posteriorly. A fluid level was present in the axillary portion of the chest at the height of the anterior portion of the third rib. The right side of the chest appeared normal.

Aspiration in the ninth space posteriorly yielded turbid, amber, odorless fluid which showed no organisms on gram stain, but a few small intracellular cocci were seen with methylene blue. Aspiration in the sixth interspace anterior to the angle of the scapula yielded foul air and pus. Operation disclosed a foul empyema overlying a pulmonary abscess about one inch in diameter, containing thick, putty-like, foul material. The abscess was unroofed.

Culture of the fluid from the empyema cavity, at operation, yielded *Staph. albus*, enterococcus and *A. israeli*. Section of the abscess wall revealed granulation tissue with purulent exudate, but no higher bacteria could be seen.

The patient received 400,000 units of penicillin daily for eleven days and 800,000 units daily for four weeks thereafter. He also received four blood transfusions, and the wound was revised twice. Two weeks after operation *A. israeli* was again cultured from the exudate in the wound. Radiotherapy was instituted, and a total of 1,200 roentgen units was administered. His temperature declined gradually over a period of four weeks and then remained normal. The wound was completely healed six weeks after operation. When the actinomyces were found in the pleural fluid, a search was made for these organisms in scrapings from the cavities of several of his teeth and from calcareous debris about the gingival margins. *A. israeli* was cultured from both locations.

COMMENT

The finding of pathogenic actinomyces, that is, the anaerobic *A. israeli*, in the pleural pus in three cases of perforated putrid lung abscess without the development of actinomycosis of the lung or chest wall is an indication of the low virulence of these organisms. In these cases the actinomyces did not behave differently from the general mixture of organisms involved in the putrid infection. In two of the cases the infection was cured by drainage alone. It is not known whether the infection would have cleared in the third case without the additional use of penicillin and radiotherapy. It is significant, however, that in the second case the infection cleared by drainage alone even though the actinomyces were present in the foul pus of an abscess in the wound sixteen months after the original thoracotomy.

The low virulence of actinomyces for the respiratory tract has also been pointed out by Kay,[8] who found these organisms in the sputum of a large number of cases of nonspecific chronic suppurative disease of the lung without any evidence of pulmonary actinomycosis.

It is a common experience that patients with bronchiectasis at first have nonodorous sputum, and that foul odor appears in the later stages as the result of the contamination of the secretions in the bronchiectatic cavities by anaerobic bacteria from the mouth. The latter do not contribute materially to the further development of the pulmonary suppurative disease in the great majority of cases, but seem to act as saprophytes. It appears that the actinomyces found in the sputum by Kay behaved in a similar manner, implanting themselves from their original habitat in the mouth into the bronchiectatic secretion without producing significant bronchopulmonary disease.

A similar course of events, but with a different time sequence, appears to have taken place in the three cases here reported. It must be concluded that the actinomyces were carried down from the mouth together with a mixture of other bacteria, including anaerobes which were responsible for the lung abscess, while the actinomyces played a passive role.

Because of the low-grade pathogenicity of the actinomyces in respect to the lungs, it appears probable that other factors are required to produce pulmonary infection by these organisms, in addition to simple inhalation. Rosebury[5] found that he could produce infection only by adding calcareous material to the culture of actinomyces, indicating that the impaction of particulate material together

with the actinomyces is a prerequisite for infection. Since this can rarely happen in the natural course of events without inhalation of other mouth bacteria, it must be concluded that these are also carried down into the bronchi at the time of the aspiration. These nonspecific bacteria certainly cause infection as easily as the actinomyces, and therefore, it seems most likely that nonspecific bacterial infection plays a role in the production of pulmonary actinomycosis, at least in its early stages.

Studies of the incidence of other types of fungi in the sputum in chronic pulmonary disease have shown that in the majority of cases the fungi act as secondary invaders, and are usually saprophytic.[11] This is such a frequent occurrence that a diagnosis of moniliasis, streptothricosis, leptothricosis, and aspergillosis is not tenable solely from the finding of these organisms in the sputum, unless other underlying diseases are entirely excluded.[10-13] A similar conclusion must be drawn in relation to the finding of actinomyces in the sputum.[8] Because of the three cases here reported the same reservation must be held even when actinomyces are found in pleural pus. A diagnosis of pulmonary actinomycosis is tenable only when fungi are found within the tissue itself.

SUMMARY

1. *A. israeli* was cultured in three cases of putrid empyema resulting from the perforation of a lung abscess. In one, this organism was also recovered from carious teeth.

2. None of the patients developed actinomycosis, although treatment in two cases consisted only of thoracotomy and drainage.

3. *A. israeli* is an organism of low-grade virulence, and is often present in the lung as a secondary invader or simply as a contaminant aspirated from the mouth and tonsils.

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USE OF THE POTTS-SMITH-GIBSON CLAMP FOR DIVISION OF PATENT DUCTUS ARTERIOSUS

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LIGATION of the patent ductus arteriosus with a single strand of nonabsorbable suture material is no longer an acceptable procedure. In a significant number of cases so managed there have been operative failures due to incomplete obliteration of the duct or its recanalization. Even with modifications of the ligation technique (use of multiple ligatures, transfixion sutures, reactive cellophane) there have been reports suggesting that recanalization may occur. The incidence of recanalization is difficult to evaluate because of the varying criteria for diagnosis and the lack of sufficient long-range follow-up. The presence of a postoperative murmur, per se, is not sufficient evidence that patency of the duct has persisted or recurred. Sometimes murmurs are audible immediately after a ductus has been completely divided. Changes in the contour of the pulmonary artery or pre-existing pulmonary artery dilatation with relative pulmonic insufficiency might account for such murmurs. The presence of other congenital anomalies must also be suspected. Consequently, a definite diagnosis of incomplete occlusion or recanalization should not be made on the basis of persistent murmur alone. If such a murmur is not typical of patent ductus in its location and character, if the murmur is present immediately following operation and does not subsequently change character, and if it is not associated with peripheral vascular signs of a systemic-pulmonary shunt, the diagnosis of recanalization requires further confirmation. However, Jones[6] has noticed a decreased incidence of postoperative murmurs since he abandoned the use of multiple tape ligatures for division and suture of the ductus, and it is well known that any artery that has been ligated in continuity shows a tendency to recanalize. On such a theoretical basis and with limited clinical evidence, it may be suspected that division of the patent ductus arteriosus is the method of choice provided the surgical mortality is not increased.

Ductus division was carried out first by Touroff in 1940.[9] In his patient division became necessary to control hemorrhage which occurred while the ductus was being isolated preparatory to its ligation. Gross was the first surgeon to divide a patent ductus deliberately. His method was reported in 1944.[4] This technique has proved eminently successful in the hands of a few surgeons. Four Crile artery forceps are applied across the isolated ductus. The two middle clamps are removed after dividing the ductus between them so that the small cuffs of vessel wall, that had been held between the jaws of the medial clamps, could be sutured with continuous rows of fine silk. In his original report Gross recommended that the jaws of each clamp be held closed by a tight rubber band

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twisted about the handles of the clamp. In a more recent report,[5] Gross stated that he had abandoned the use of rubber bands, closing each clamp to the first ratchet. Crafoord[3] has applied clamps temporarily to the aorta above and below the ductus and has then divided the ductus while the aortic stream was completely interrupted.

Our experience with these two techniques of division has been limited. During our first attempt to divide the ductus by Gross's method, the Crile clamps, closed with rubber bands, slipped from position on either end of the ductus. Bleeding from the aorta and pulmonary artery was controlled with difficulty, but division and suture were completed and the immediate postoperative course was uncomplicated. Ten days after operation the patient developed classical signs of pulmonary embolism. There was no evident site for thrombus formation and, consequently, it was postulated that thrombosis had occurred at the site of ductus suture. The patient recovered and has done well, but in view of these complications and the relatively infrequent opportunity to perfect this method of division, we reverted to a technique of obliterating the duct with two ligatures of umbilical tape with an intermediate transfixion suture of heavy braided silk. Since April, 1947, we have used the method for division which will be described here and which has been mentioned in a previous publication.[2]

The high incidence of spinal cord lesions in dogs after complete acute clamping of the aorta, even of short duration, has deterred us from using Crafoord's procedure deliberately. Our only experience with clamping of the aorta during ductus division occurred at operation on a 9-year-old girl in 1946. The original plan was to perform double tape ligation, but during the dissection there was profuse hemorrhage from a tear in the posterior wall of the ductus. The aorta was clamped completely, proximal to the ductus, for periods of twenty-two minutes and thirteen minutes, with an interval of twenty-five minutes between, in order to control hemorrhage while the ends of the duct were sutured following its division. There was no evidence of leg paralysis after this prolonged period of aortic obstruction, and the postoperative course appeared uneventful, although a routine electrocardiogram on the tenth postoperative day showed T-wave inversion in the lateral precordial unipolar leads. This finding suggested a small area of myocardial ischemia although there was no evidence of infarction and the patient has continued to do well. The T-wave changes have disappeared. We are unable to explain such an occurrence and were unable to duplicate such an electrocardiographic change during clamping of the aorta in the dog.[10]

There are a number of factors that have contributed to the problem of devising an entirely satisfactory technique for division of a ductus. The average duct is a very short vessel with a large diameter. This makes simple division between ligatures extremely hazardous. The ligatures are likely to roll off the ends of the divided vessel, particularly at the aortic end where the large lumen is exposed to high systemic arterial pressure. Experience with pulmonary resections has demonstrated adequately that a single ligature, with a transfixion suture added to eliminate the danger of the ligature rolling off the vessel end, provides complete security for closure of the proximal end of a divided pulmonary artery. At its pulmonic end the wall of the duct has essentially the

same characteristics as that of the pulmonary artery. This end of the duct is exposed only to the relatively low blood pressure of the pulmonary artery after division of the ductus has been accomplished. Ligation and transfixion provide a satisfactory method for closure of the pulmonic end of the ductus unless the vessel is exceptionally large. The wall of the duct at its aortic end, on the other hand, is like the wall of the aorta; it is relatively thick and friable so that a simple encircling ligature tends to crush and to split the intima. The intimal damage which occurs with crushing of the aortic end of the ductus predisposes to recanalization when ligation techniques are used. Exposed to the high systemic arterial pressure, even a transfixion suture is likely to be dislodged from the aortic end of the duct and, should such a suture produce a small tear in the aortic wall, hemorrhage may become progressively severe and uncontrollable.

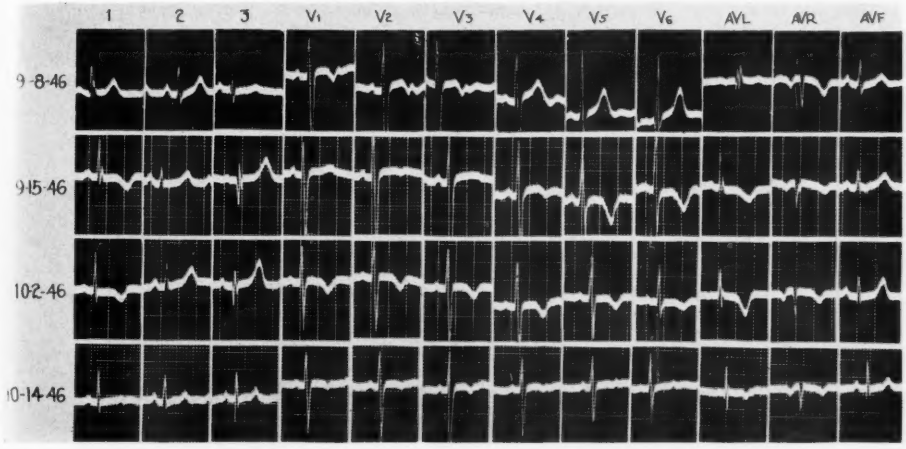

Fig. 1.—Electrocardiographic changes following complete clamping of the thoracic aorta distal to the left subclavian artery during division of patent ductus. The aorta was completely clamped for periods of twenty-two and thirteen minutes with an interval of twenty-five minutes between the periods of clamping. Preoperative electrocardiogram (Sept. 8, 1946) showed upright T waves in the unipolar precordial leads, V_4, V_5, and V_6. Operation was performed on Sept. 9, 1946. The first postoperative tracing was recorded on the sixth postoperative day and showed inversion of the T waves in V_4, V_5, and V_6. T-wave inversion regressed over a period of five weeks after operation.

The posteromedial wall of the ductus at its pulmonic end may be closely adherent to the left main bronchus. By the usual blind approach for dissection of this part of the ductus, it is often difficult to free sufficient length for division by ligature or suture techniques. The ductal wall is fragile here and is likely to be torn, especially if it has been weakened by superimposed bacterial infection.

Methods requiring the application of clamps to the ductus itself have many disadvantages: (a) danger of slipping or dislodgment of the clamps, (b) too small a cuff available for closure, (c) inclusion of thin ductal tissue in the aortic closure with the consequent danger of late aneurysm, (d) unsuitable for the very short ductus where four clamps cannot be applied, (e) trauma of the vessel wall close to the area of suture predisposing to rupture or aortopulmonary fistula.*

*A new clamp devised by Potts[3] grasps the ductus with prongs without crushing it and avoids some of these disadvantages, particularly (a) and (e).

When the technique for creation of a side-to-side anastomosis between the pulmonary artery and aorta was reported by Potts, Smith, and Gibson[7] it seemed that the aortic clamp described by them could be used as well to divide a ductus as to create one artificially. Consequently, the following procedure was developed which we feel offers the advantages of complete aortic obstruction without the dangers inherent in complete interruption of aortic blood flow.

OPERATIVE METHOD

In order to obtain adequate exposure and enough room for placing the Potts clamp on the aorta, a posterolateral incision is considered obligatory. The patient is placed on the operating table in the right lateral decubitus position. An endotracheal tube is used for the administration of cyclopropane and ether in a closed circle system. An ankle vein is cannulated routinely and a continuous slow infusion of dextrose solution maintained during the operation. Blood is available but is usually not required. The fourth rib is resected from behind its angle to the anterior axillary area and the pleural space is entered through the periosteal bed of the resected rib. It is particularly important that the rib resection be carried well medial to the angle of the rib posteriorly to facilitate manipulation of the aortic clamp. Occasionally it has been advisable to resect a small posterior segment of an adjoining rib to increase exposure.

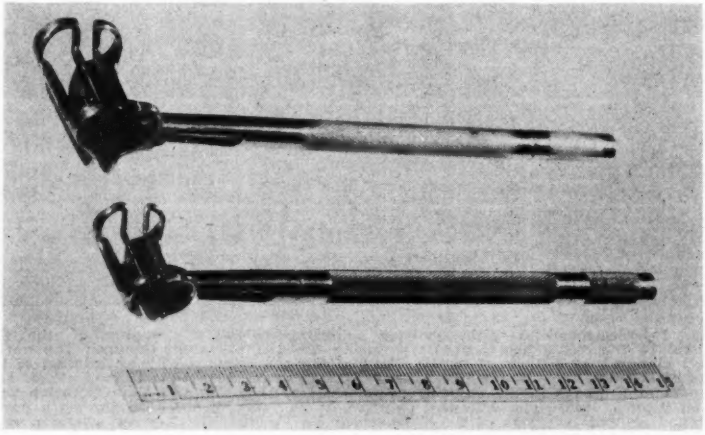

Fig. 2.—Aortic clamps of the type described by Potts, Smith, and Gibson. The lower clamp is the standard size described by them. The upper clamp was made locally for us. The diameter of the portion encompassing the aorta and the length of the jaw is one-third larger than the standard clamp dimensions.

The aortic clamp is of the type described by Potts and his associates.* The standard clamp used by Potts is too small to be used easily in any but the smallest patients considered for ductus operation. Our largest clamp was made locally and is one-third larger than the standard Potts clamp. The standard and larger clamps are shown in Fig. 2.

Mediastinal dissection is started by incising the pleura over the first part of the descending aorta from the origin of the left subclavian artery to a point about 2 cm. distal to the ductus. A side extension of this incision, passing directly over the ductus, may prove desirable later in the dissection. The aorta is exposed by subpleural dissection and the segment opposite the ductus orifice is mobilized. It may be necessary to divide one or more intercostal arteries and smaller branches of the aorta passing to the mediastinum.

*Available from V. Mueller & Company, Chicago, Ill.

Fig. 3.—Incision of the mediastinal pleura over the first part of the descending aorta. This incision is more posterior than the usual incision for ductus ligation.

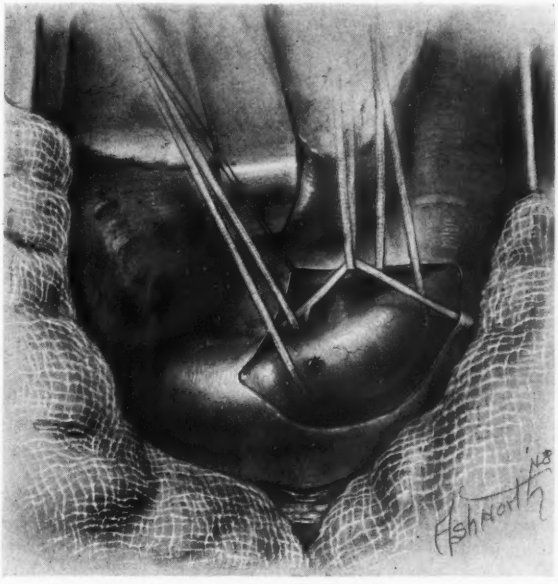

Fig. 4.—Dissection of the aorta and ductus is completed. Umbilical tapes are placed around the aorta above and below the ductus. The vagus nerve is retracted anteriorly showing the recurrent laryngeal nerve passing medial to the ductus. One intercostal artery has been divided.

Care must be taken that such vessels are not sheared off close to the aorta. Division between ligatures is recommended rather than division between clamps. One must avoid injury to the thoracic duct while dissecting the aorta away from the thoracic vertebrae. If this vessel is traumatized, ligation is essential to prevent chylothorax.

When the aorta is adequately mobilized, strips of umbilical tape are passed around it above and below the ductus. Gentle traction with these tapes exposes the posterior aspect of the aortic end of the ductus so that dissection can be completed preparatory to placing the aortic clamp. The recurrent laryngeal nerve is mobilized so that it will not be included in the jaws of the aortic clamp. The Potts clamp is then placed around the aorta and

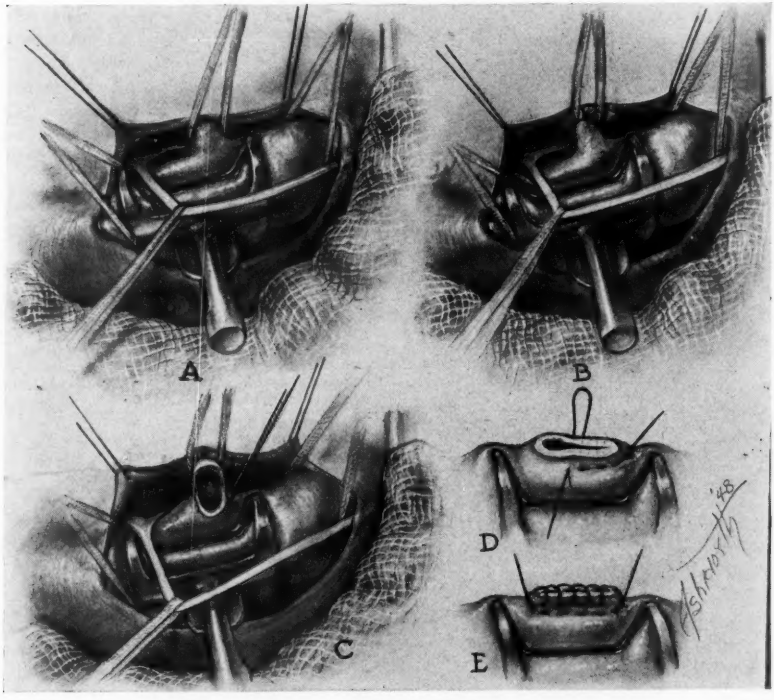

Fig. 5.—Details of the division and suture.
A, The clamp isolates the ductus from aortic blood flow but allows blood to pass into the descending aorta.
B, The pulmonic end of the ductus is ligated and transfixed as close to the pulmonary artery as possible.
C, The initial artery stitch is anchored at the upper angle of the aortic end of the ductus. The ductus is then divided. For purposes of clarity in this drawing, the vagus nerve is retracted to the aortic side of the ductus incision although in practice it is necessary to retract it toward the ligature on the pulmonic end of the ductus. Less tension is placed upon the vagus nerve than is shown.
D, The aortic end of the divided ductus is repaired with a continuous mattress suture of artery silk. Intima is approximated to intima.
E, The mattress suture is overlayed with a continuous glover's stitch.

slowly closed to occlude the aortic end of the ductus. The encircling tapes may be used to hold the aorta in position within the clamp during its closure. As soon as the clamp is completely closed the continuous thrill over the pulmonary artery and lung is eliminated. Appearance of a systolic thrill over the aorta distal to the clamp indicates that blood flow through the aorta has not been completely interrupted. The blood pressure in the arms may show a variable rise.

When the anesthetist has determined that latent cyanosis has not become evident as a result of eliminating blood flow through the ductus, isolation of the duct is completed. The dissection is greatly facilitated by early mobilization of the aorta and occlusion of the aortic end of the ductus. After elevating the aorta carefully by means of the clamp, the thin posterior wall of the ductus can be freed from the left main bronchus under direct vision.

The pulmonic end of the ductus is ligated with heavy braided silk and transfixed with medium braided silk. The ductus is then divided between the pulmonic ligature and the aortic clamp. The vagus and recurrent laryngeal nerves should be retracted to the pulmonic side of the point of sectioning to avoid injury. These nerves are then allowed to fall between the divided ends of the duct where they will not interfere with closure of the aortic end. The aortic clamp is elevated and rotated to present the divided end of the duct. This is repaired with a continuous horizontal mattress suture of 00000 Deknatel on a curved atraumatic needle which approximates intima to intima. The first stitch of this

Fig. 6.—Operative field after division.

suture is placed above the proximal angle of the proposed line of division and close to the proximal jaw of the Potts clamp. By placing and tying this stitch before the duct is divided, bleeding which might occur from leakage around the proximal portion of the clamp is obviated. The running mattress suture should conform to the curve of the aortic wall avoiding creation of an appreciable pouch in the aortic wall. The closure is reinforced with a running glover's stitch over the divided end of the duct and in the cuff created by the mattress stitch. The aortic clamp is released slowly. If appreciable bleeding occurs, the clamp can be retightened and additional sutures placed. Oozing through the points of needle puncture may occur but will subside spontaneously.

During the operation the anesthetist inflates the collapsed left lung at intervals. Warm isotonic saline solution is used to keep the pleural surfaces moist. Unless marked tachycardia is present, 3 to 5 c.c. of 1 per cent procaine solution are injected about the left vagus nerve early in the operation. Following a final irrigation of the pleural cavity with

warm isotonic saline solution, the incision in the mediastinal pleura is loosely approximated with interrupted stitches and the chest wall closed in layers while lung expansion is maintained with water suction through a catheter placed in the wound. This catheter is removed during the closure of the skin. No drains are used.

We have used this method for division of the patent ductus arteriosus in eleven patients. Early in the series one patient succumbed at the time of operation as a result of hemorrhage. The hemorrhage is attributable to faulty technique. Lacking a Potts aortic clamp of sufficient size to completely isolate the aortic end of the ductus, the lip of a very large and dilated aorta, which bulged from between the jaws of the clamp, was occluded by compressing it with a loop of umbilical tape drawn tightly against the jaws. As a result of this ill-advised maneuver the aortic wall was split, the tear extending rapidly in a proximal direction so that all attempts to suture it were of no avail. We would like to stress the importance of using a clamp of appropriate size in each case, believing that the tragic outcome in this case might thereby have been avoided. No mishap has occurred during or following surgery in the other ten patients and all have remained well. Six of these patients had a soft systolic murmur over the left upper chest following operation. There has been no change in the character of these murmurs and there have been no other physical findings suggesting recanalization. In one of the six patients an isolated stenosis of the left pulmonary artery was found just distal to its ductal orifice and this may be the source of the persistent murmur. There has been no apparent correlation between postoperative murmurs and anatomic evidence of pulmonary artery enlargement.*

DISCUSSION

Use of the Potts-Smith clamp for division of patent ductus arteriosus provides certain advantages despite the more extensive dissection of mediastinal structures required. Mobilization of the aorta has allowed more freedom in the development of deep tissue planes about the ductus and in freeing the fibrous bands attached to the posterior wall of the ductus. During dissection of the thin-walled duct a measure of satisfaction can be taken from the knowledge that the aorta is mobilized for temporary clamping in case of hemorrhage. After the aortic clamp is closed, the dissection can be completed without the danger of spontaneous rupture from full aortic pressure within the unsupported walls of a thin ductus, one whose walls are damaged by bacterial endarteritis, a ductus

*Since preparation of this manuscript, ten additional patients have had ductus division by this technique. One more patient died of hemorrhage. In this case operative findings suggested marked pulmonary hypertension, though pulmonary artery pressure was not measured. The left pulmonary artery and the ductus were both enormous in caliber; nevertheless, placement of the aortic clamp and isolation of the duct were readily accomplished. While ligating and transfixing the pulmonary end of the duct, prior to dividing it, the pulmonary artery tore and the patient became exsanguinated before intrapericardial isolation of the pulmonary artery, and its temporary occlusion with a Blalock clamp, could be completed. While ligation and transfixion will control the pulmonary end of the duct satisfactorily in most instances, it is evident that a vessel of large caliber will be crushed and subject to tear when obliterated by a surrounding ligature, especially when ligation is performed close to junction with a large tense vessel like the pulmonary artery in this case. Plastic suture of the pulmonary end of the duct, as well as the aortic, is advisable in some cases. Potts has devised ductus clamps[3] with prongs which grasp and occlude the vessel without crushing and without danger of slipping. In our most recent case, with a large short ductus, both ends of the duct were sutured using a Potts ductus clamp on the pulmonary end and a Blalock modification of the Potts aortic clamp[1] on the aortic end during division and suture. The operation proceeded very smoothly. The latter clamp, being narrower at its back than the original design of Potts, is more readily fitted to the aortic end of the duct, particularly when the origin of the left subclavian artery is close to that of the ductus arteriosus.

with aneurysmal dilatation. The period of temporary clamping while the dissection is completed allows considerable time for the development of latent cyanosis in the patient with unsuspected pulmonic stenosis, although we have seen no case of this type where the diagnosis could not be made without operation. With a clamp of the Potts design, there is little danger of slipping since the aorta is encircled. Any pulsation is transmitted equally to the encompassing jaws. Inspection of aortic segments occluded by the clamp at operation and in the animal laboratory reveals no evidence of aortic damage. Occasionally a small subadventitial hemorrhage will be seen along the site of application, but there has been no evidence of trauma to the media or intima.

Because of the peculiar trough shape of the portion of the clamp isolating the ductus, a sizable segment of aorta is excluded for suture. Consequently, a double row of sutures can readily be placed along the aortic end of the ductus at its junction with the aorta. The contour of the clamp should also allow its use for division of very short ductuses, where the technique of clamping the ductus proper cannot be used. A portion of aortic intima and ductal lumen is available for inspection. Evidence of endarteritis may be demonstrable and the sutures may be placed accurately in the aortic wall proper. Both ends of the ductus can be repaired in such a way that no significant vascular pouch will remain at the side of the aorta or pulmonary artery.

Experience with side-to-side anastomosis of the aorta to the pulmonary artery indicates that great care must be taken in suturing the aorta to avoid tearing of the friable vessel wall. Of all the great vessels, the aorta seems particularly subject to tearing, and to us this hazard has seemed greater in suturing longitudinal incisions than transverse incisions, as in the resection of aortic coarctation.

The major disadvantage of this method has been that it is sometimes difficult to place the clamp so that its jaws completely encircle the aorta proximal to the ductus. This problem is encountered when the origin of the ductus is more proximal than usual so that the clamp must be placed at a point where the curvature of the aortic arch is prominent. Placement of the clamp is particularly difficult when fusiform dilatation of the aorta extends proximal to the ductus, when the aorta is large and relatively inelastic as in older age groups, and when the origin of the left subclavian artery is close to that of the ductus. Careful dissection of the deeper surfaces of the aorta above the ductus minimizes this disadvantage, but occasionally, even with adequate dissection, considerable manipulation has been required to place the clamp in position. A clamp that is adequate in size to encircle the dilated aorta or subclavian origin and has been modified to fit the medial curve of the aortic arch should eliminate this difficulty.* Death of our second patient is ascribed to the use of a clamp which was too small to encircle a dilated aorta. The excessive manipulation required produced tear-

*Since preparation of this manuscript, the article of Freeman, Leeds, and Gardner (Surgery 26: 103, 1949) came to our attention. They have modified the Potts aortic clamp to overcome certain difficulties encountered in its use for ductus division. The Blalock modification, as mentioned in a previous footnote, also appears to have definite advantages.

ing of the aorta. However, with increasing experience, we feel that the increased control and exposure available during dissection of the ductus proper will compensate for any additional risk.

SUMMARY

1. A technique for division of patent ductus arteriosus is presented. The Potts-Smith-Gibson clamp, designed for the creation of aortopulmonary shunt by suture, has been adapted to the reverse procedure, division of patent ductus arteriosus.

2. Application of the procedure is reported.

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THE ROLE OF ESOPHAGEAL MOTILITY IN THE SURGICAL TREATMENT OF MEGA-ESOPHAGUS

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THE ACTUAL underlying physiopathology of mega-esophagus is still a matter of uncertainty in spite of much work done and many theories set forth in an attempt to understand the problem. There have existed three great schools of thought regarding the pathologic physiology of mega-esophagus. According to the theory of achalasia[1-5] which has been generally accepted by many modern workers,[6-8] mega-esophagus is due to a lack of relaxation of the cardiac sphincter which normally occurs[9] when each peristaltic wave conveying food through the esophagus reaches it. In support of this theory are the findings of Rake,[10] Cameron,[11] Beattie,[12] Etzel,[13] Lendrum,[14] and others who have demonstrated degenerative changes in the ganglion cells of Auerbach's (myenteric) plexus. When the myenteric plexus is partially or wholly destroyed, such as was done by Tamiya and Tamura,[15] the esophagus becomes irregularly active and later quiet, and the cardia fails to open normally in response to an oncoming bolus of food. On the basis of experimental work and as a result of clinical and post-mortem observations, Etzel[16] came to the conclusion that the degeneration in human beings is due to a chronic vitamin B_1 deficiency. The achalasia theory explains that the disease is produced by disturbance in the nerve-muscle mechanism of the cardia. This is dependent upon degenerative changes in the vagal nerve supply which result in an inability of the circular muscle to relax due to preponderance of sympathetic control.

Knight[17] has concluded from his experiments on the cat that there exists a true functional esophageal sphincter at the cardia. A condition resembling cardiac achalasia[17-19] can be produced in some animals by complete or partial bilateral vagal section, which is relieved by subsequent division of the appropriate sympathetic fibers.[17] Furthermore, simultaneous division of the sympathetic nerves prevented this condition.[17] Following the double denervation there occurs eventually a normal movement of food after temporary disturbances.[17] On the basis of these experimental findings division of the sympathetic nerves has been carried out for cardiac achlasia in man with some promising results[20-23] but certainly not always with relief to the patient,[24] so that it seems that it is still better to dilate the esophagus and later, as necessary, to cut the sphincter in a manner such as by esophagogastrostomy of the Finney type. There is a wide origin of the sympathetic fibers which supply the esophagus,[25, 26] so that some of the failures of sympathectomy may have been due definitely to incomplete sympathetic denervation of the affected esophagus.

Among the many other theories, Mikulicz[27] and Huss,[28] as well as others, popularly contended that the dilatation of the esophagus occurred secondary to a true spasm of the cardia. Carlson, Boyd, and Pearcy[29] confirmed and extended

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Openchowski's[30] observations on the reflex control of the cardia and lower esophagus, which laid the groundwork for the possible existence of cases of clinically true cardiospasm. However, because of the poor results obtained by various surgeons[31-34] after bilateral vagotomy for idiopathic mega-esophagus, it was generally agreed that this procedure is not justified because its rationale is apparently based on a false premise.

The third group, including Sauerbruch and von Hacker,[35] Jackson,[36] and Hill[37] among others, widely advocated a phrenospasm as the underlying cause of the condition. However, the observation of obstruction above and below the diaphragmatic crura appears to rule out the possibility of their malfunction as a cause of the symptomatology.[38] Because gastrointestinal difficulties sometime follow unilateral phrenicectomy, Ballon, Wilson, Singer, and Graham[39] investigated the condition of the esophagus and stomach after this operation. In the two cases of mega-esophagus to be reported here, the obstruction also occurred above the diaphragmatic crura. Furthermore, in spite of arrest of movement of the left leaf of the diaphragm following phrenic crush during transthoracic exploration, the esophageal hiatus, which was normal and more than adequate in size to transmit the constricted cardiac end of the esophagus, did not increase in size as might be expected if the so-called cardiospasm were due to a phrenospasm. Division of the diaphragmatic crura[40, 41] and phrenicoexeresis[42] were accomplished by many but with unsatisfactory effects, so that these procedures have been generally discontinued as independent methods of treatment of mega-esophagus.

To the surgeon the problem of the underlying pathologic physiology of achalasia is a matter of importance. Many surgeons, including Ochsner and DeBakey,[43] have noted that even after adequate esophagogastrostomy the esophagus frequently does not return to its normal size, as the stomach returns to a normal size following the surgical relief of hypertrophic pyloric stenosis. Clagett and his associates[44] stated that although good functional results are obtained, retrogression in size of the dilated esophagus after esophagogastrostomy should not be expected. The problem of the underlying physiopathology of achalasia is further important since, we are convinced, the late postoperative clinical end results, even though good, are frequently not as pleasing when the esophagus remains atonic and dilated.

Recently we had an unusual opportunity to study this problem since we had noted similar phenomena following adequate esophagogastrostomy, as determined by satisfactory relief of symptomatology, by the barium swallow with Trendelenburg position test, and by postoperative esophagoscopy. Because methods of determining the functional behavior of various parts of the esophagus, heretofore, have been so equivocal and inadequate, we have felt that esophageal motility studies by the balloon and kymograph method may be of help in solving this problem. We have made esophageal motility studies of three normal persons under varying conditions and of two patients following esophagogastrostomy in the treatment of esophageal achalasia. These data indicate that the intrinsic motility of mega-esophagus varies greatly, being often irregular, uncoordinated,

and ineffective, and that it may be entirely absent, thus disclosing that a disturbed peristalsis or even total motor paralysis of the whole esophagus is present. We now wish to present the clinical and esophageal motility studies together with a detailed correlation between these two phases of the work.

EXPERIMENTAL METHODS

An easily distensible and markedly strong balloon was attached to an Einhorn tube in the usual manner. In order to determine accurately by fluoroscopy and by x-ray film studies (Fig. 1) the position of this balloon in the esophagus, we coated its inner lining with a radiopaque material, Rugar.

Fig. 1.—The position of the Rugar-lined balloon in the esophagus was easily determined by fluoroscopy and x-ray film study. In this patient (O. P.) with achalasia, as for normal persons, the balloon could be inflated only slightly without resulting in pain. A deglutition effect, which was less than normal, was almost constantly present, as measured by an almost continuous pull on the Einhorn tube. Esophageal fluid collected over the top of the balloon when the latter was held in one position during an experiment.

After placing the balloon in the desired position, the tube was connected to a bromoform-water manometer (Fig. 2). The balloon was never inflated to such an extent that it acted as an obstructing agent but rather as the usual bolus of solid food. In order to keep the distance from the incisor teeth to the balloon constant during any one experiment it was necessary to anchor the Einhorn tube securely to the patient's face with adhesive. This was done to counteract the constant pull on the Einhorn tube due to the constant attempt, by the muscular contraction and peristalsis of the normal esophagus, to force the balloon onward into the stomach. Observations were made in each person at three levels of the esophgus; the upper esophagus, above the aortic arch; the middle of the esophagus; and the lower esophagus, just above the diaphragm.

At the beginning of each experiment the balloon was inflated to the desired pressure of bromoform. The time and motility were recorded by a slowly revolving long kymograph. The inkwriters for the electric automatic timer, which recorded every ten seconds, and for the

manometer (Fig. 2) were composed of Monel metal, were sensitive, and recorded beautifully in ink* the time and the motor activity of any part of the esophagogastrointestinal tract.[45] The apparatus (Fig. 2) is easily assembled and much less fragile than former recording apparatus. With this apparatus the recording of motility is as easy as the recording of the basal rate of metabolism by the Benedict-Roth apparatus.

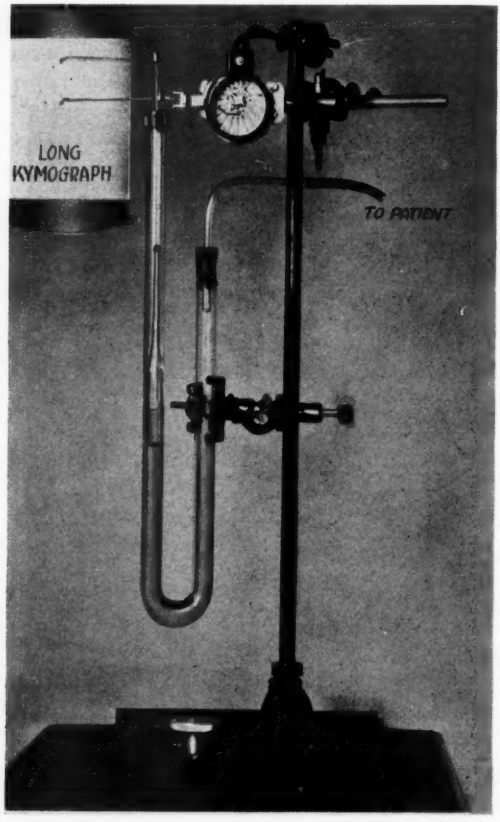

Fig. 2.—Apparatus useful for motility studies of the digestive tract. The inkwriters for the electric automatic timer and for the manometer are composed of Monel metal. The apparatus is easily assembled and much less fragile than former motility recording apparatus.

Each experiment was made in the postabsorptive state under basal conditions. Information obtained by barium swallow and by the observed clinical effects upon the inflated balloon was complementary to that obtained from the kymograms. The data considered together gave adequate conceptions of the action of the musculature of the esophagus.

ESOPHAGEAL MOTILITY STUDIES OF MEGA-ESOPHAGUS

The clinical, radiologic, and esophagoscopic progress, as well as the esophageal motility of two patients with mega-esophagus, was studied during a period of from one and one-half to

*The paper and ink were obtained from Warren E. Collins, Inc., Boston, Mass.

two and one-half years following esophagogastrostomy of the Finney type. The protocols may be reviewed briefly as follows:

Protocol of O. P., No. 460072.—The patient, a white schoolgirl, 17 years of age, first became aware of difficulty in swallowing in 1937, at 9 years of age. She was known as "graveyard" at 15 years of age because she was almost five feet tall and weighed only 50 pounds. Because of her symptoms she followed a very poor and capricious dietary regimen. She had had carious teeth for years.

Physical examination disclosed the patient to be well developed but emaciated and asthenic. She was five feet two inches in height but weighed only 80 pounds. There were many markedly carious teeth. Most of the reflexes were moderately hyperactive.

The usual laboratory tests were negative. Fluoroscopy as well as x-ray film studies (Fig. 3, *A*) of the esophagus during barium swallow on Oct. 4, 1945, revealed typical evidence of achalasia. Radiography of the colon revealed it to be atonic with a tendency to megacolon.[47-49] Esophagoscopy on Oct. 6, 1945, showed the esophageal mucosa to be of a normal pink color. The cardia at times could be seen to relax but never enough to admit the scope. During withdrawal of the scope, areas of spastic constrictions were seen to come and go along the esophagus. These were so marked that they produced a tight gripping of the scope both on introduction and withdrawal, indicating actual spasm. Alvarez[46] theorized that this phenomenon is most likely due to the pathologic changes in the myenteric plexus which led to the uncoordinated irregular contraction of the esophageal musculature. These procedures revealed no evidence of organic obstruction.

Conservative treatment for achalasia using a well-balanced diet, psychotherapy, antispasmodics, and frequent dilatation with an Einhorn dilator was tried for three months, with only temporary and partial benefit. Occasionally the patient could keep down an entire moderately large meal. While eating she would feel the esophagus fill up until she vomited approximately one cupful of undigested food. There was accompaning pain in the region of the larynx severe enough to cause lacrimation. By drinking water and forcing it down she was able to force solid foods into the stomach. She frequently became very hungry. There was no gain in weight.

She therefore entered University Hospital on Jan. 3, 1946, when a transthoracic cardioplasty was made, following a technique similar to that for Finney's pyloroplasty, using an inner tier of chromic catgut and an outer tier of interrupted Halsted mattress sutures of fine black silk. Four finger tips could be inserted easily through the new stoma. Usual reconstruction without drainage was carried out.

The esophagus was found to be greatly dilated throughout its whole length until it reached the cardiac end where it suddenly became constricted to about 1 cm. in diameter just above the crura and continued in this constricted fashion through the esophageal hiatus. There was no evidence of muscular enlargement at the cardia. There were no other local gross evidences of abnormality of the cardia. The esophageal hiatus was more than adequate in size to palpation for the passage of the esophagus. A phrenic crush was done and the esophageal hiatus was again palpated but was found not to have changed appreciably in size or shape. The disease in this one patient would appear not to have been due to a phrenospasm.

The postoperative course was smooth. She was discharged on the twenty-first postoperative day on a regular diet. Subsequently she suffered none of the former symptoms. Her general health was greatly improved. She had gradually gained weight to the present 93 pounds.

Protocol of M. J., No. 465143.—The patient, a colored man, 26 years of age, entered University Hospital on July 23, 1946. He sustained the first attack of vomiting and dyspnea ten years previously. The vomitus consisted of undigested food which frequently had a sour taste and at times a foul odor. During the three to four years before admission vomiting had become more severe and occurred at least once after each meal. Nausea seldom was associated. A feeling of fullness of the chest and palpitation often followed a meal. There had been a loss of about fifteen pounds during the three months prior to admission.

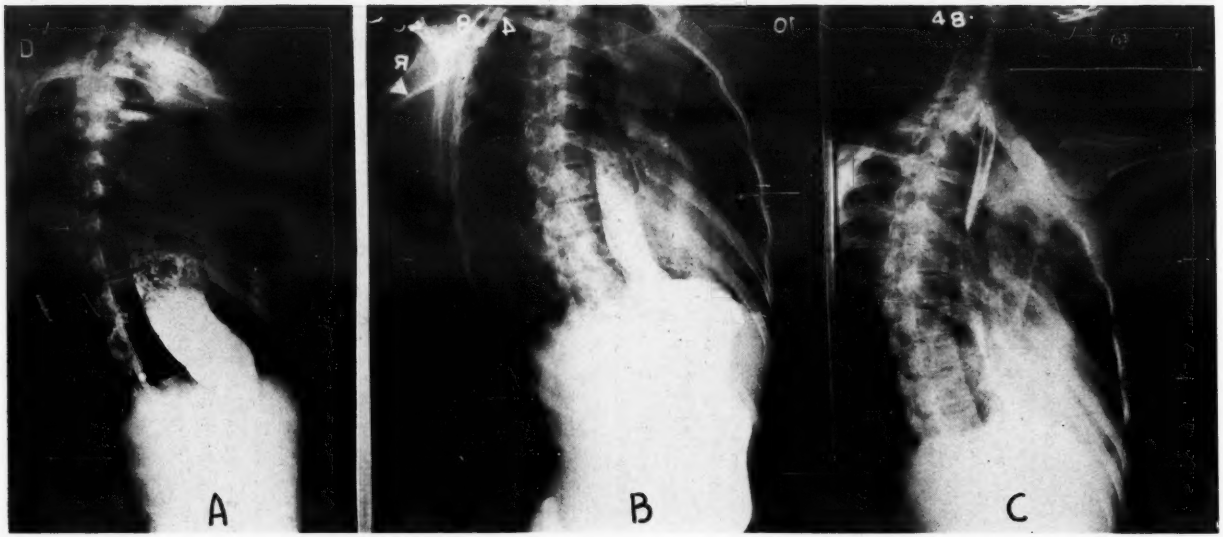

Fig. 3.—*A*, X-ray film studies during barium swallow on Oct. 4, 1945, showed practically no barium passing through the narrowed cardiac end of the esophagus. There was a marked fusiform dilatation of the esophagus above the cardia. *B*, Same case, three months postoperatively; showed definite decrease in size of the esophagus and great increase in emptying of the barium into the stomach. *C*, Same case, two and one-half years following a Finney type of cardioplasty. This was the nearest return of the esophagus to normal clinically and physiologically that we had observed following operation. Roentgenography on July 22, 1948, showed the esophagus to have a normal diameter. There remained no fluid level in the esophagus. In fact, this view with thick barium was difficult to obtain because of rapid and complete emptying.

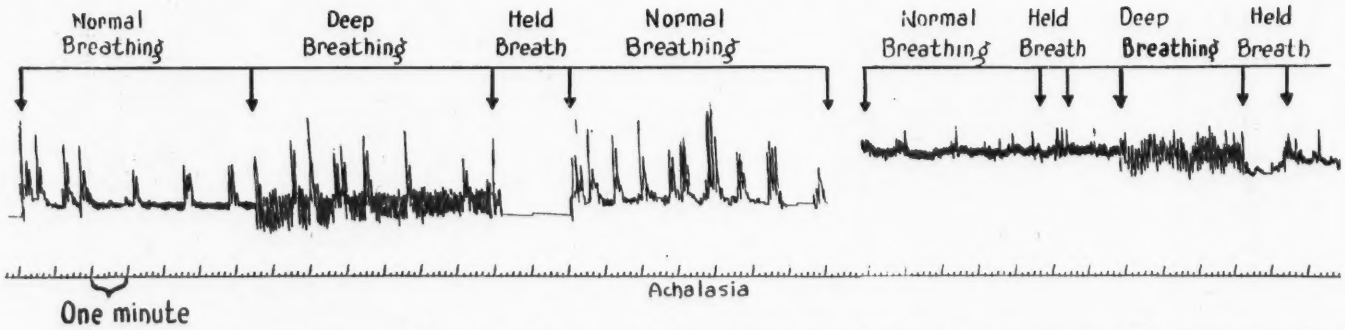

Fig. 4.—Tracing of esophageal motility of achalasia (O.P.) taken on May 8, 1948. During this period of infrequent and irregular contractions of low amplitude the strength of deglutition was minimal. In contrast to normal, the character of the breathing played a definite part in changing the character of the tracing. When the breath was held, there was at times no evidence of intrinsic motility, and at other times the contractions were irregular and shallow.

Physical examination disclosed a cheerful, colored man. He was well developed but undernourished. The teeth were carious. The reflexes were for the most part hypoactive.

The routine laboratory tests were negative. Radiographic study of the chest (Fig. 5, *A* and *B*) showed the typical evidences of achalasia of the esophagus. Esophagoscopy revealed a pale granular mucosa. The esophagus was tremendously dilated, tortuous, and in folds so that examination was not entirely satisfactory. No organic basis of obstruction of the cardia was found.

Conservative measures, including repeated attempts at dilatation with an Einhorn dilator were unsuccessful and uncomfortable. It was therefore felt advisable to treat this intractable achalasia by a transthoracic esophagogastrostomy.

The usual preoperative measures were followed, including use of penicillin, sulfasuxadine, daily lavage of the esophagus, a liquid diet, and adequate additions of the usual vitamins. Under general anesthesia, using Pentothal Sodium intravenously for induction and cyclopropane, ether, helium, and oxygen intratracheally, a transthoracic cardioplasty of the Finney type was carried out, using an inner tier of catgut and two outer tiers of fine black silk. Four finger tips could be inserted through the new stoma. Penicillin was used locally. Stomach attachments, repair of the diaphragm, and closure of the chest wound were made in the usual fashion. Drainage was not used.

The pathology was merely an exaggeration of that found in O. P. At the constricted area there was no hypertrophy of the muscularis layers and no evident pathology of the mucosa. The muscularis layers as well as the mucosa of the hugely dilated portion of the esophagus were greatly thickened.

The postoperative period was entirely uneventful. Soon after discharge on the fourteenth day he began to eat a regular diet. He gained twenty-five pounds in body weight during the first year after operation.

RESULTS AND DISCUSSION

Three normal persons served as controls. They were studied under varying conditions over many hours. The character of the intrinsic motility of the normal esophagus varied greatly not only from person to person at the same level of the esophagus but at various levels and at various times at the same level in the same person. Normally, however, changes in the character of the breathing produced no appreciable change in the type of tracing (Fig. 6, *upper*) when the intrinsic motility was vigorous enough to result in good deglutition effect.

In the normal esophagus, the balloon could not be distended with over 20 to 40 c.c. of air, the amount depending upon the location of the balloon and the type of person, without producing pain. We agree with Jones[55] that sudden distention of a local segment of the esophagus was much more likely to produce a painful sensation than the more gradual introduction of air through the tube into the balloon. In our patients, also, the localization of the discomfort was usually sharply defined substernally and corresponded fairly closely with the actual level of the balloon. Most subjects complained of an uncomfortable fullness in the upper portion and more frequently of a burning sensation as the balloon was inflated at the lower levels of the esophagus. These sensations were sometimes localized in the back and rarely in the neck or face.

Normally there occurred almost a persistent intrinsic esophageal motility which was elicited by a nonpain-producing inflated balloon held in place by adequate anchorage of the tube. This balloon therefore acted as a bolus of food.

Fig. 5.—*A*, Radiogram of chest showed a diffuse marked widening of the mediastinum. At the level of the clavicles there was a definite fluid level. *B*, Film studies during barium meal swallow showed a colossal sigmoid mega-esophagus. Only a small amount of the ingested barium trickled into the stomach. *C*, Radiographic studies seventeen months following esophagogastrostomy (Finney type) showed slight decrease in the size of the esophagus but not more than after adequate decompression of the esophagus before operation. The esophagus was still markedly dilated and an esophageal fluid level persisted in spite of a wide-open stoma. *D*, Same case, two and one-half years following operation showed same findings as in *C*.

The motility was active, responsive to stimulation, well coordinated, and effective. It produced powerful peristaltic waves and an excellent deglutition effect which was noted as a strong, persistent pull on the Einhorn tube even when the subject was placed in Trendelenburg position. This tracing (Fig. 6, *upper*) was typical of the lower esophagus (34 cm. from the incisor teeth) of R. E., a normal subject 40 years of age, as taken on Dec. 31, 1947. The balloon was slowly inflated with 30 c.c. of air, which was the maximum which could be introduced without causing pain. The shallow notches superimposed on the intrinsic contractions represent transmitted respiratory (so-called esophageal respirations) and vascular (so-called esophageal pulsations) movements.

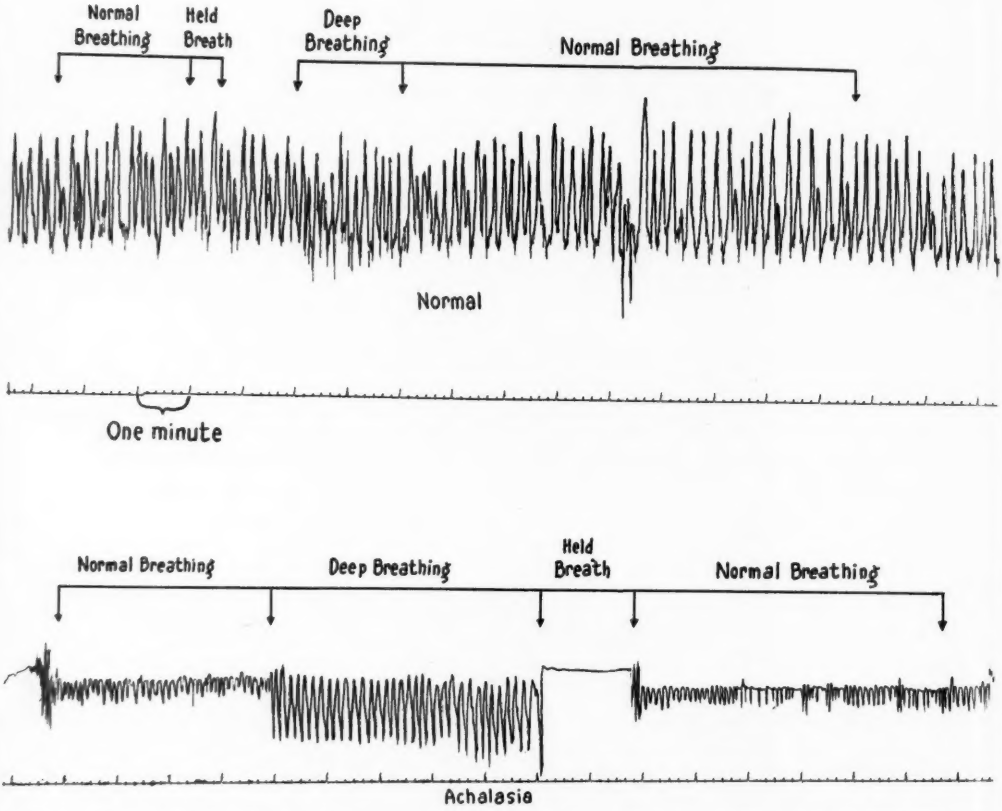

Fig. 6.—Tracings of esophageal motility of a normal person (*upper*) and of a patient with achalasia (*lower*).

The tall, regular, and frequent contractions of the normal esophagus (*upper*) produced powerful peristaltic waves and a strong deglutition effect. Changes in character of the breathing produced no appreciable change in the character of the intrinsic motility of the esophagus.

In marked contrast to normal, the esophagus (M.J.) of achalasia (*lower*) showed continuous absence of intrinsic motility and no deglutition effect was produced as measured by absence of a detectable pull on the Einhorn tube. This tracing represents esophageal respirations which are synchronous with and due to the respiratory movements of the chest. When respiration was held there were no esophageal movements.

The number of contractions which occurred normally varied greatly from one every three minutes over a short duration to an average of seven every minute over long periods of time.

The amplitude of the normal contractions also varied greatly from short periods of feeble ones of less than 5 mm. of bromoform to long periods of tall ones averaging 7.5 cm. in height. The contractions of frequent occurrence and of vigorous character were accompanied by the strongest deglutition effect.

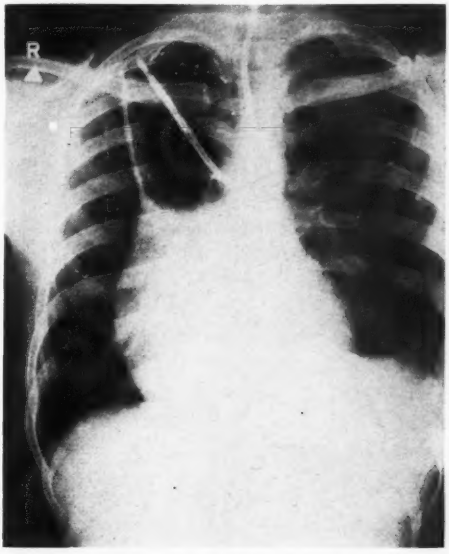

Fig. 7.—Achalasia (M.J.). When the patient was in the upright position, no matter in what portion of the esophagus we placed the balloon it always floated to the top of the esophageal fluid just as the balloon was inflated. This was in marked contrast to the normal and to O.P. No deglutition effect was produced on this balloon even when it was inflated to the point of substernal pain and well against the wall of the hugely dilated esophagus. This necessitated introduction of 500 c.c. of air.

The administration of atropine sulfate, $\frac{1}{150}$ gr. subcutaneously, to normal subjects produced in a short time a definite period of relative quiescence with a great decrease in the number and height of contractions. A decrease in the deglutition effect accompanied. Administration of urecholine, 5 mg. subcutaneously, to a normal person produced a diametrically opposite effect.

We operated on the two patients with mega-esophagus included in this study, not as an initial procedure but only after so-called conservative measures, such as psychotherapy, medical therapy, including vitamins and antispasmodics, and repeated dilatation of the cardia by a dilator had failed to give adequate relief after a reasonable trial. A type of esophagogastrostomy was employed similar to Finney's pyloroplasty and the technical end result of the operation is illustrated in Fig. 8. As explained previously, observations during operation revealed no evidence that the mega-esophagus was due to a phrenospasm.

The clinical result was good for both patients. Vomiting no longer occurred after operation; the dysphagia, if present, was minimal, and the chest pain and dyspnea disappeared. Both patients gained body weight quite rapidly and maintained a good nutritional balance. The presence of an adequate stoma in each case was demonstrated by the good clinical end results as well as by esophagoscopic observations. Yet, peristaltic deglutition as observed by use of the fluoroscope revealed this function of the esophagus much more active, coordinated, and responsive to stimulation for one patient (O.P.) so that it was difficult to obtain a good film of the esophagus (Fig. 3, *C*) because of the rapid emptying of thick barium on July 22, 1948, two and one-half years following operation. Similar fluoroscopic studies showed the other mega-esophagus (M. J.) was atonic, and

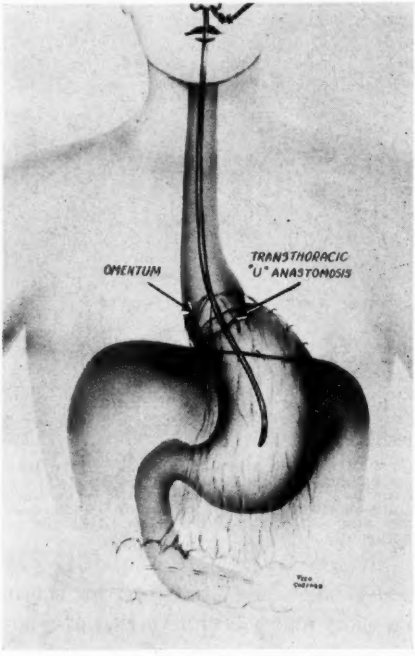

Fig. 8.—Representation of the operative end result.

unable to evacuate its contents properly, so that an x-ray film (Fig. 5, *D*) on Feb. 1, 1949, two and one-half years after operation, still showed the presence of an 18 cm. esophageal fluid level. Furthermore, over the same length of time postoperatively, the esophagus gradually returned to a normal size by July 22, 1948, in one patient, O. P. (Fig. 3, *C*), while the hugely dilated esophagus of M. J. (Fig. 5, *D*) did not appear to reduce in size appreciably by Feb. 1, 1949, over its decrease in size following adequate preoperative decompression. The latter phenomena have been noted by many surgeons[43, 44] following what seemed to be an adequate cardioplasty in many cases of mega-esophagus and even after suc-

cessful dilatation of the cardia. Jackson[56] stated that symptomatic relief is obtained by dilatation, but the associated diffuse mega-esophagus is permanent.

To our knowledge the latter phenomena have not been adequately explained. The problem of the underlying physiopathology of achalasia is further important since, we are convinced, the late postoperative functional end results on careful inquiry are frequently not as pleasing when the esophagus remains atonic and dilated. For instance, the patient (M. J.), who had the persistent mega-esophagus postoperatively, complained of inability to eat rapidly because on rapid eating there developed a sensation of nausea and of temporary holdup of food in the esophagus. The esophageal contents slowly emptied into the stomach, and it was only through slow eating that he was able to remain symptomless and to reach and maintain a normal nutrition. The other patient (O. P.), who obtained postoperative clinical, physiologic, radiologic, and esophagoscopic end results practically approaching the normal, was able to eat rapidly without symptoms. Much thought has also been given to the question as to whether mega-esophagus represents in some cases a complete motor paralysis of this structure. We have, therefore, made extensive investigations of the esophageal motility of two patients with achalasia and have compared these findings with those obtained of the normal esophagus.

We have made several motility studies, each lasting a few hours, at various times from twenty-two to twenty-six months after adequate esophagogastrostomy for O. P. The balloon was inflated with 20 c.c. of air, which was the most that this patient would tolerate without resulting in substernal pain. They showed almost continuous periods of irregular contractions of variably low amplitude and infrequent occurrence (Fig. 4). *Esophageal respirations* were prominent in this achalasia tracing (Fig. 4).

The number of contractions (O. P.), with the inflated balloon held in constant place and acting as a bolus of food, varied from one every three minutes over a short duration to four every minute over long periods, the average being one-half the normal.

The amplitude of the contractions (O. P.) also varied greatly but not as much as normal. The contractions varied from a few very shallow waves to frequent ones of an average of 2.5 cm. (bromoform) tall. Their amplitude averaged one-third the normal. As in normal persons, contractions of more frequent occurrence and of greater amplitude were accompanied by a better deglutition effect.

The administration of atropine sulfate, $\frac{1}{150}$ gr., subcutaneously, to O. P. abolished the intrinsic activity to a great extent just as for the normal esophagus. During this period the active deglutition effect was practically abolished. Administration of urecholine, 5 mg. subcutaneously, produced a diametrically opposite effect with establishment of vigorous contractions which produced so much pain on contracting over the inflated balloon that the air had to be released, and the balloon was taken into the stomach.

We made several studies of the movements of the esophagus over many hours, seventeen to twenty-nine months following adequate esophagogastrostomy for M. J. They showed complete absence of the motor function of the esophagus

(Fig. 6, *lower*) and no peristaltic activity was produced as measured by the absence of a detectable pull on the Einhorn tube carrying the inflated balloon. When the patient was in the upright position, no matter in what portion of the esophagus we placed the balloon it always floated to the top of the esophageal fluid just as the balloon was inflated (Fig. 7). This is another evidence of the absence of peristalsis of the mega-esophagus of this patient. These findings are in sharp contrast to the normal or to the findings (Fig. 1) in the other case (O. P.) of achalasia presented herewith. *Esophageal respirations,* which are synchronous with and due to the respiratory movements of the chest, are outstanding (Fig. 6, *lower*). They vary, as is readily appreciated (Fig. 6, *lower*) with the character of the respiration and are due to changes in the degree of the negative intrathoracic pressure. With increasing inspiration there occurs an increasing negative intrathoracic pressure, an increasing inflation of the balloon, and an increasing inrush of air from the manometer closed system into the balloon and a production of the lowest level of excursion on the kymogram at the point of deepest inspiration (Fig. 6, *lower,* deep breathing). Certain patients with achalasia unknowingly use these mechanics as an aid in overcoming the increased resistance at the cardia by taking a deep breath, placing the chin on the chest, and increasing the intrathoracic pressure and thereby the intraesophageal pressure by a forced expiratory effort against the closed glottis and closed cricopharyngeal sphincter which work normally in achalasia, as shown by others as well as by our cases. Contrary to these findings, it was felt heretofore that peristalsis occurred normally in the colossal mega-esophagus[54] but that it was ineffective in deglutition because the bolus of food was not large enough to be grasped by the hugely dilated esophagus.

When the patient is placed in Trendelenburg position after barium swallow, the barium rushes freely into the esophagus from the stomach in the patient in whom the function of the cardia is interfered with by resection of the esophagogastric junction as for carcinoma of the lower esophagus or by a Finney type of cardioplasty as for esophageal achalasia. This backward rush is suddenly stopped by the cricopharyngeus sphincter so that regurgitation does not occur. This condition offered an admirable opportunity to observe the almost continuous and presumably normal esophageal peristalsis in a patient 75 years of age who was symptomless two and one-half years after I had performed a radical esophagogastrectomy with a low esophagogastrostomy repair in the treatment of a squamous-cell carcinoma of the lower esophagus. These studies revealed that normally true peristalsis of the esophagus consisted of a wave of contraction passing along the esophagus at a fairly rapid speed. This wave was strong and frequently propelled the heavy barium contents uphill before it for 5 to 8 cm. from the cricopharyngeus and at times for the whole length of the esophagus. Thus it emptied completely the esophagus of its barium contents as the contraction proceeded. There occurred no detectable leakage of barium into the contracted area of the esophagus until the contraction wave came to a stop, at which time relaxation of the whole esophagus allowed the barium to rush again to the cricopharyngeus and the process was repeated. These peristaltic waves occurred more frequently and were more effective when the patient attempted to swallow

voluntarily. The peristaltic waves of the esophagus did not conform to the classic description of peristalsis as set forth by Bayliss and Starling.[57-59] The wave of contraction was not preceded by a detectable wave of active relaxation and enlargement as concluded by Bayliss and Starling to receive the onflowing radiopaque barium. This conforms to the recent work of McLaren[60] who reached conclusions similar to ours after the ingenious use of roentgenkymography during barium swallow with the patient upright. Nor did suction action of the third phase or the squirting action of the pharyngeal phase of swallowing[50-53] play an appreciable role in esophageal swallowing normally when the patient was in Trendelenburg position. Normally, the propulsion of esophageal contents is therefore dependent to a great extent upon an effective peristalsis.

Fig. 9.—The bolus of thick barium (*A*) being swallowed by M.J. while in acute Trendelenburg position took a normal course to enter the esophageal pool (*B*) without spillage from the pool into the pharynx.

Further inquiry into the movements of the esophagus in achalasia in a patient (O. P.), in whom postoperatively the esophagus appeared to return to near normal clinically, radiologically, and esophagoscopically, revealed an irregularity and a great decrease below normal in the strength of the peristaltic waves. In the Trendelenburg position the barium contents rushed rapidly against the cricopharyngeus sphincter. The wave of contraction was weak and usually produced a feeble undulation of the esophagus. The barium contents were never propelled uphill before it for over 2 to 3 cm. and this occurred only when attempts were made to swallow voluntarily. The barium contents were never detectably emptied into the stomach during Trendelenburg position.

In a patient with achalasia (M. J.) who received less anatomic benefits from esophagogastrostomy than O. P., there occurred at no time discernible peristalsis. The barium meal, which collected against the cricopharyngeal sphincter during

Trendelenburg prison, lay there like water in a stagnant pool. This is similar to the decreased motor activity of the stomach which is at times seen following a vagotomy.[62] This lack of peristaltic deglutition to aid Magendie's classical third stage of swallowing indicates widespread vagal paralysis of the esophagus. It may represent an underlying primary physiopathologic mechanism; yet it could constitute a secondary alteration of the physiology of the whole muscular tube based on an advanced complicated stage of the disease. Interestingly enough, barium swallowed by M. J. while in acute Trendelenburg position took a normal course to enter the esophageal pool of barium without detectable spillage from the pool into the pharynx (Fig. 9). These motility and radiologic findings in achalasia are similar to the findings which I shall publish later[64] of two other patients with achalasia studied preoperatively.

Our patient, M. J., with achalasia sustained definite pain when a segment of the esophagus was stretched by an inflated balloon. It is reasonable to believe this another indication that the sympathetic innervation remained intact.[63] These data contradict the earlier consensus that pain in achalasia is conveyed by vagal afferent fibers as the problem was reviewed in 1930.[61]

These data suggest that we revise present views concerning the extent of the esophagus involved in achalasia. They indicate that the condition is not localized necessarily to the cardiac end of the esophagus, as is frequently stated by many authorities, but that functional abnormalities may often involve the whole length of the esophagus. These findings emphasize that our views of the extent of the cardiac operation must be better formulated as well as the postoperative treatment of the late functional condition improved in certain cases in an attempt to obtain a more normal functional end result.

SUMMARY AND CONCLUSIONS

1. Three normal persons served as controls. They were studied under varying conditions over many hours. These studies show that organized frequent contractions of good amplitude are related normally with effective active peristalsis of the esophagus and play an outstanding role in Magendie's third stage of swallowing.

2. For two cases of intractable achalasia of the esophagus, a type of transthoracic esophagogastrostomy was employed similar to Finney's pyloroplasty. Observations during operation revealed no evidence that the mega-esophagus was due to a phrenospasm.

3. One of the patients with achalasia showed the nearest return of the esophagus to normal clinically, physiologically, radiologically, and esophagoscopically that we have observed following esophagogastrostomy. Motility studies disclosed almost continuous periods of irregular contractions of infrequent occurrence and variably low amplitude. The contractions were not well organized, so that minimal strength of peristalsis was produced as measured by minimal pull on the Einhorn tube carrying a balloon inflated lightly against the wall of the esophagus. However, the contraction waves had sufficient power to force the balloon into the stomach and the deglutition effect was strong enough to produce

very good emptying of the esophagus through the large operative stoma when the patient was in the upright position. On the other hand, emptying of the esophagus was not appreciable in the Trendelenburg position. As in normal persons, contractions of more frequent occurrence and of greater amplitude were accompanied by a better deglutition effect.

4. Another patient with achalasia gained about as much amelioration of symptoms following esophagogastrostomy and yet the hugely dilated esophagus decreased but slightly in size over a period of two and one-half years following operation, a condition which has been noted by other surgeons. Radiologically, the esophagus was unable to evacuate properly its contents so that an esophageal fluid level persisted. Motility studies showed continual absence of the motor function of the esophagus, and no peristaltic activity was produced as measured by the absence of a detectable pull on the Einhorn tube.

5. Patients whose function of the cardia was abolished by operative procedures offered an admirable opportunity to observe the movements of the esophagus for the reason that when placed in Trendelenburg position after barium swallow the barium rushed freely into the esophagus from the stomach to be stopped suddenly by the cricopharyngeal sphincter so that regurgitation did not occur. The normal movements of the esophagus of a patient thus studied two and one-half years following excision of a carcinoma of the lower esophagus frequently emptied the esophagus of its barium but did not conform to the classic description of peristalsis, since the wave of contraction was not preceded by a wave of active relaxation. In achalasia the movements were uncoordinated or absent and the strength of the contraction wave was greatly decreased below normal or absent, so that the esophagus in Trendelenburg position was never appreciably emptied of its barium.

6. Information obtained by barium swallow and by observed clinical effects on the inflated balloon which acted as a bolus of food was complementary to that obtained from the esophageal motility studies. The data considered together gave adequate conceptions of the action of the musculature of the esophagus. These data indicated that the intrinsic motility of mega-esophagus varies greatly, being often irregular, uncoordinated, hyporesponsive to stimuli, and not completely effective. In fact the intrinsic motility may be entirely absent and completely fail. This differs from that of the organic obstructive lesions of the esophagus in which there occurs usually a normal motility or a hypermotility but rarely even a hypomotility, depending upon the degree of obstruction and the stage and complications of the organic disease. Achalasia of the esophagus also differs from true esophagospasm in which there may occur a hypermotility.

7. A disturbed peristalsis or even total motor paralysis of the whole esophagus may be present in this disease. The latter is the underlying factor which explains the inability of the esophagus to return to normal size following an adequate esophagogastrostomy. It may also explain some of the failures of this operation. Our studies indicate that the stoma must be made sufficiently large and constructed in such a manner as to offer the least resistance to gravitational onflow of the esophageal contents into the stomach since the factor of peristaltic force is here diminished or even abolished.

8. Our data indicate that the disease is not localized necessarily to the cardiac end of the esophagus as is commonly believed, but that functional abnormalities may often involve the entire length of the esophagus.

9. It seems irrational to use a member of the atropine series in the treatment of true achalasia since atropine sulfate produced a further depressant effect on the esophageal motility and peristalsis. Atropine, however, does decrease the amount of salivation which may result in symptomatic relief. Our data indicate that a sympathicolytic drug such as ergotamine or Priscol and a drug which has the peripheral effects of acetylcholine, such as urecholine, are worthy of a reasonable clinical trial in the conservative treatment of achalasia and in certain cases in the adequate postoperative treatment of the patient.

10. Normal accuracy of localization of the inflated balloon is generally present in achalasia so that the sensory supply presumably remains undisturbed. Recent data contradict the earlier consensus that pain in achalasia is conveyed by vagal afferent fibers.

11. These newer observations offer further evidence for and are compatible with the general theory of achalasia as an underlying mechanism of cause of idiopathic mega-esophagus.

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PERMANENT FILLING OF DEAD SPACE IN THE PLEURAL CAVITY AFTER PNEUMONECTOMY

An Experimental Study

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THE purpose of this experimental study was to test whether a lightweight, plastic prosthesis, roughly the size and shape of the lung, could be used to occupy the dead space left in the pleural cavity after removal of one lung. A prosthesis was placed in the pleural cavity after right or left pneumonectomy in a series of twenty-one dogs. Postoperative studies included roentgenograms of the thorax and, in a few experiments, exploratory thoracotomy. Several dogs were killed while they were under anesthesia in order that reaction to the prosthesis might be investigated in detail. The first dogs in which the prosthesis was used have now gone nine months since operation. Because we were unable to study pleural reaction and exudate as thoroughly as we wished in dogs, especially during the early postoperative period, a similar series of experiments was carried out in a series of rats. This report, however, deals with the results of our study on dogs. A report of the study on rats will be made later by one of us (J. R. R.), and only the pertinent conclusions of the experiments on rats will be included in this report. In the sense that the experiments on dogs must be continued for a much longer period and that the prosthesis is being modified and improved, the present report is preliminary.

It was not within the scope of our study to investigate physiologic function of the remaining lung after pneumonectomy, or to compare its function when no prosthesis was used with function when a prosthesis was used in the empty pleural cavity. The possibility that respiratory function is impaired is suggested by the fact that permanent anatomic changes occur in the thorax after the removal of one lung and a segment of the phrenic nerve on that side. Narrowing of the intercostal spaces and rise of the diaphragm constrict the pleural cavity to a significant extent after the lung has been removed, the residual space is usually filled by the heart and other mediastinal structures, and the remaining lung usually enlarges to a degree necessary to compensate for the mediastinal shift. This enlargement of the lung has been called "compensatory emphysema," although it has been shown by Rienhoff, Reichert, and Heuer[1] and by Behrend and Mann[2] that this is not true emphysema in dogs. They found that increases in size and weight of the lung were the result of the fact that the lung was more fully expanded and contained more blood than normally. The possibility remains, however, that simple distention of the lung may eventually lead to emphy-

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sematous changes in some persons. Evidence concerning the function of the lung, whether it be simply distended or pathologically distended, has been accumulated in recent years.

Cournand and Berry[3] as well as Birath and co-workers[4] have concluded that overdistention of the remaining lung after pneumonectomy reduces the breathing reserve, produces an increased tendency to dyspnea, and allows marked reduction in the oxygen saturation of arterial blood to occur during strenuous exertion. Such changes were somewhat less likely to occur if pneumonectomy was performed during childhood or early adolescence and more likely to occur in older adults than in young adults.[3, 5] Thoracoplasty as a second operation reduced the degree of overdistention in adult patients and improved ventilatory efficiency. Similarly, prevention of overdistention in a patient who already had emphysema before pneumonectomy, by placing air or oil in the thorax on the side of pneumonectomy, caused improvement in pulmonary function.

Another method of prevention of overdistention of the remaining lung was reported on recently by Johnson and co-workers.[6] In a preliminary report of studies on dogs they described the use of a prosthesis to occupy the empty pleural cavity after pneumonectomy. In one series of dogs observed for as long as four months, an airtight hollow box of zirconium measuring 10 by 6 by 3.5 cm. was used. In another series, hollow balls of methyl methacrylate 1 inch (2.5 cm.) in diameter, similar to those used by Wilson[7] for an extrapleural pack, were used to fill the pleural cavity. Dogs of the latter group were observed as long as two months. A significant degree of pleural reaction was observed in dogs with the zirconium prosthesis, but only minimal pleural reaction occurred when the plastic balls were used.

Our study was the outgrowth of one in which we used gelatin foam sponges to occupy the dead space after pneumonectomy in dogs and rats.[8] Although this material was gradually absorbed, postoperative shift of the mediastinum and gross pleural effusion were prevented. It was noted also that dogs so treated were much less dyspneic and considerably more active after operation than dogs in which gelatin foam sponge was not used. These observations suggested that function of the remaining lung was not impaired but improved, during the postoperative period, by a foreign material placed in the empty pleural cavity and encouraged us to investigate the possibilities of use of a nonabsorbable prosthesis.

We believed the most suitable prosthesis would be one which would fit the pleural cavity and leave a minimum of dead space, would be just large enough to prevent mediastinal shift, and would not produce harmful pleural reaction or other ill effects. Our desire to have the prosthesis leave as little dead space as possible and to avoid having empty spaces around the prosthesis, such as are present between balls, was based on theoretic objections that dead space would permit motion, friction, mechanical trauma, and discomfort, and that the greater the dead space the greater the volume of free fluid and the greater the danger from infection.

METHODS

At the time the study was undertaken we[9, 10, 11] had had two years of experience with Polythene (polyethylene) in various living tissues and were impressed that it caused

neither inflammatory nor foreign body reaction in many tissues and that it was not altered by exposure to living tissue. Accordingly, it seemed that Polythene might be suitable for the thoracic prosthesis. We constructed a roughly lung-shaped bag with Polythene film (thickness 0.005 inch) by fusing the edges together with heat and pressure (Fig. 1). The bags contained air and, in order to give them the desired shape, rather tightly packed fiber glass. In flat view the cephalic end of the bag was rounded but narrow, while the caudal end was wide but sloping, so that the posteroinferior portion of the bag in situ was lower than the anteroinferior portion. The bags were slightly thicker at the base than at the apex. The density of the prosthesis was appreciably less than that of inflated lung. After the bags had been tested for leaks they were sterilized by immersion in 1:1,000 aqueous solution of benzalkonium chloride (Zephiran chloride). The inside of the bag and its contents were not sterilized.

Fig. 1.—The Polythene bag prosthesis. The outer layer of the prosthesis is Polythene film (thickness 0.005 inch), the edges of which have been sealed together by heat. The prosthesis contains air and rather tightly packed fiber glass.

The prosthesis was placed in the pleural cavity of the dogs, after right or left pneumonectomy, before the thorax was closed. A sufficient number of sterile prostheses of various sizes was always available so that one of suitable length, width, and thickness was on hand. No attempt was made to aspirate the remaining air after the thorax was closed, and thoracentesis was not performed during the postoperative period. In judging the size of the prosthesis to be placed in the thorax we allowed for the rise of the hemidiaphragm and narrowing of the intercostal spaces and tried to select a Polythene bag which would be the size of the pleural cavity during maximal expiration after such changes had taken place. Our early experience with Gelfoam taught us the danger of filling the pleural cavity to such an extent that function of the remaining lung was compromised.

RESULT OF EXPERIMENTS

Right pneumonectomy was performed on 10 dogs and left pneumonectomy on 11 dogs; each of these dogs had a Polythene bag prosthesis placed in the pleural cavity after removal of the lung. All operations were performed with the dog under ether anesthesia and with aseptic surgical technique. No difference was observed between dogs in which the right lung was removed and those in which the left lung was removed. All dogs tolerated the operation well and were in excellent condition during the early postoperative period.

Six of the 21 dogs of the series died between four and fourteen days (average seven and one-half days) after operation. Necropsy revealed that in each instance the bag had split open at a seam and that acute empyema was present. Another dog died twenty days after operation because of fibrosis around the inferior vena cava near the right atrium. A loosely attached thrombus occluded the entire thoracic part of the inferior vena cava, the liver was greatly enlarged and nearly black in color, massive abdominal ascites was present, and both pleural cavities contained massive amounts of blood stained fluid. The cause of the fibrosis about the vena cava and thrombosis could not be determined with certainty, but we believe it was mechanical trauma produced by the prosthesis. The bag was too small for this dog and could have rotated enough so that during the respiratory cycle the seam along the posterior border scratched back and forth on the cephalic end of the inferior vena cava. Another dog died on the twenty-eighth postoperative day because of erosion of the left internal mammary vein and the massive hemorrhage which followed. There seemed to be no question that in this case the seam along the apex of the bag had impinged on the vein and finally lacerated it. Two other dogs of the series died, 1 at five months and the other at six months postoperatively; the cause of death in each instance was acute empyema caused by a leak in the bag and exposure of its nonsterile contents to the pleural cavity. The leak in one bag consisted of a burst seam, in the other of a crack at the site of a wrinkle.

Two of the 11 dogs that lived were killed, while they were under ether anesthesia, in order that pleural reaction could be studied, 1 dog four and one-half months and the other seven months after operation. Both dogs appeared to be in excellent general condition and weighed more than they weighed before operation. The heart and mediastinum were in the normal position in the midthorax, and the remaining lung in each dog appeared entirely normal, both grossly and histologically. The right pleura (side of pneumonectomy) in the dog killed at four and one-half months was almost everywhere of normal thickness and appearance (Fig. 2). Slight thickening was noticed in a few areas of the parietal pleura, the principal area being over the healed operative wound. This side of the thorax contained 30 c.c. of clear straw-colored fluid. There were a few erythrocytes in the fluid, and the leucocytes numbered 2,890 per cubic millimeter, of which 5 per cent were lymphocytes and 95 per cent were neutrophils. The Polythene bag prosthesis was not completely intact. The fiber glass at the apex of the bag was stained green and was slightly moist, apparently the result of a small leak that allowed some fluid to enter the nonsterile interior of the prosthesis.

The pleural surface on the left, the side of pneumonectomy, in the dog killed at seven months likewise showed few changes. The mid-portion of the pericardial pleura, however, was pink and granular, and there were two similar pink, granular areas in the mid-parietal region, each about 3 cm. in diameter. The pleura in the region of the thoracotomy wound was opaque and gray but glistening. The remainder of the pleural surface appeared normal, and this side of the thorax contained 20 c.c. of clear, straw-colored fluid. A tiny hole was present in the corner of a wrinkle near the lower end of the prosthesis, and the air that had been in the bag was completely replaced by clear, straw-colored fluid. Although the inside of the prosthesis had not been sterilized when it was made, significant bacterial growth obviously had not occurred.

Nine dogs of the series are living and all appear to be in excellent general condition. Two have had the prosthesis in place for nine months, 2 for eight months, 2 for seven months, 2 for six months, and 1 for five months. Five of these dogs had right pneu-

monectomy and 4 left pneumonectomy. Exploratory thoracotomy was performed in 1 dog at two months and in another at three months; biopsy of the mediastinal and parietal pleurae revealed slight thickening. The gross appearance of the pleura in each dog was normal. In 1 dog 20 c.c. of free fluid was found in the pleural cavity and in the other not enough fluid was present for aspiration. The fluid in the 1 dog was pink, probably because of contamination by fresh blood.

Roentgenographic examination on the day after operation was made in 5 dogs. Except for slight narrowing of intercostal spaces, slight rise of the hemidiaphragm, and absence of the usual pulmonary markings, the roentgenogram was that of a normal thorax. A detectable amount of fluid was not present, and the mediastinum was in the normal position. Roentgenograms of the thorax were obtained on all the dogs at about two or four weeks and thereafter at intervals of one or two months. In general, the examination

Fig. 2.—Dog killed four and one-half months after right pneumonectomy and implantation of a Polythene bag prosthesis in the right pleural cavity. The mediastinum is in normal position and the right pleural surface is slightly thickened. The lower medial portion of the right pleural cavity shows the 30 c.c. of fluid which were present at the time of necropsy.

disclosed no further change for the first four to five months. After this time the prosthesis, instead of being invisible roentgenographically, had a mottled appearance. Later, some of the prostheses became radiopaque. For a time, this finding confused us. We suspected at first that a capsule might have formed about the prosthesis and then became calcified. The dogs which were killed gave us the explanation of this finding. The roentgenogram, made just before death, of the dog killed at four and one-half months, showed mottling at the upper end of the bag; the fiber glass at this end of the bag was found to be moist. The roentgenograms of the dog killed at seven months had shown mottling of the bag at five months, and at the time of his death the bag was radiopaque. Necropsy showed that the bag was filled with fluid. We have concluded, therefore, that radiopacity of the prosthesis indicates fluid which has entered through a leak.

RECENT EXPERIMENTS

Because we learned from the series of 21 dogs that the Polythene bag prosthesis had structural weakness, that is, a sharp seam along its edge and a tendency to break open, we have discarded this prosthesis. We are continuing our study* and are now employing a hollow, air-filled, methyl methacrylate (Lucite, Plexiglass) prosthesis. The prosthesis has a perfectly smooth external surface, is very light in weight, and is molded to fit the pleural cavity. Two dogs have had this prosthesis in the left pleural cavity for two and one-half months, another for two months, and still another for one month. No deaths or complications have occurred thus far, and the mediastinum appears, on roentgenographic examination, to be in the normal position.

COMMENT

Our study has demonstrated that a prosthesis roughly the size and shape of the space remaining in the pleural cavity after right or left pneumonectomy will prevent the occurrence of mediastinal shift and, presumably, overdistention of the remaining lung. We are aware, however, that our study has been merely a test of whether a prosthesis would prevent mediastinal shift and that physiologic studies must be made to determine whether the prosthesis is effective in protection against the harmful effects of overdistention of the remaining lung. Whether our single-unit prosthesis has advantages over the multiple hollow-ball prosthesis described by Johnson[6] remains to be determined. We question, however, that it would be more difficult surgically, as Johnson feared, to remove a large single-unit prosthesis, should the need arise, than a number of small balls. If the balls become fixed in position some of them might be hard both to find and to remove. We feel that the single-unit prosthesis, shaped to fit the pleural space, might have certain advantages over the multiple-ball prosthesis. For example, the patient might have fewer subjective symptoms because there would be no other foreign body on which the prosthesis could impinge. Also, it seems to us an advantage to provide the pleural cavity with a preformed prosthesis and a theoretic disadvantage to let uncontrollable forces collect and stabilize a number of small objects into an effective prosthesis. We suspect, furthermore, that there are dangers inherent in the small-ball prosthesis; namely, that a ball might insinuate itself into a mediastinal pocket and produce pressure on the esophagus or a large blood vessel, or that the organic material (fluid or tissue) which fills the spaces between balls, especially spaces most distant from a source of blood supply, might be prone to infection. Finally, there may be danger of overfilling the pleural cavity and applying pressure to the mediastinum if balls are used because the surgeon may be tempted to fill the pleural cavity completely and not to follow directions based on preoperative measurements.

The Polythene bag prosthesis, like the multiple, hollow, methyl methacrylate ball prosthesis of Johnson,[6] produced minimal pleural reaction as long as it did not leak and allow pleural fluid to come in contact with the nonsterile contents of the prosthesis. Our observations of pleural reaction, however, are based on only 4 dogs, of which 2 were explored before the prosthesis had begun to leak, 1 dog was killed when a leak had started, and 1 was killed when the prosthesis was full of fluid. In order to obtain more information concerning pleural reac-

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tion than we could on a series of dogs we made a study of pleural reaction in rats. Careful determination of volume and cellular content of pleural fluid and histologic examination of the pleura were made in a large series of rats killed at frequent intervals ranging from one day to several months after pneumonectomy and implantation of a Polythene bag prosthesis. These studies showed that the prosthesis did not cause formation of excessive or abnormal pleural fluid and that pleural reaction was limited to the formation of a very thin fibrous capsule about the prosthesis. In our dogs, such a capsule has not been observed.

We recognize the structural defects of the prosthesis we have used in this study but we are confident the defects can be corrected. The molded, hollow, methyl methacrylate prosthesis that we have used lately appears to be structurally stronger than the Polythene prosthesis, although it is much more rigid; it is superior also in that it has no protruding seams. We believe that the best permanent prosthesis will be one having the general shape of the space it is to occupy, will be of just the right size to prevent appreciable mediastinal shift, will have permanent structural strength, will be as nonirritating as the prosthesis we have used, and will have so smooth a surface that there will be no danger of mechanical trauma to neighboring living tissue.

SUMMARY

A lightweight bag, made by heat-sealing Polythene film and filled with fiber glass and air, roughly the shape of the pleural cavity, and of sufficient size to prevent mediastinal shift, was placed in the pleural cavity after right or left pneumonectomy in a series of twenty-one dogs. Only the outside of the prosthesis was sterile. During the early postoperative period all dogs were in excellent condition.

Six dogs died during the first two weeks because of acute empyema caused by rupture of the prosthesis. Two dogs died, five and six months after operation, respectively, from the same cause. Two dogs died from lesions attributed to mechanical trauma of blood vessels adjacent to the prosthesis.

Two dogs were killed, one at four and one-half months and the other at seven months. The mediastinum of each was in normal position, and the pleura showed a minimal degree of thickening.

Nine dogs are well and have had the prosthesis in place for periods ranging from nine to five months. The position of the mediastinum has always been normal in these dogs.

Eventually a leak consisting of either a crack or a rupture of the Polythene covering developed in all the Polythene bag prostheses. This has permitted the air within the prosthesis to be partially or completely replaced by fluid. Although sterilizing the inside of the prosthesis and its contents would probably lessen the danger of bacterial growth in the prosthesis and of empyema, this weakness of the Polythene bag makes further use of it as a prosthesis undesirable unless the defects are corrected. Recent experience with a lightweight,

hollow, molded one-piece prosthesis of methyl methacrylate in a series of four dogs indicates that this prosthesis is superior to the one made with Polythene and filled with air and fiber glass.

This study has demonstrated that the pleural cavity tolerates a large, nonirritating prosthesis after removal of the lung and that mediastinal shift can be prevented by such a prosthesis. This study and a similar one in rats have demonstrated that the prosthesis does not cause formation of excessive amounts of fluid and that it causes minimal pleural reaction.

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THE ROENTGENOLOGIC MANIFESTATIONS OF PARASTERNAL OMENTAL HERNIA

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THE preoperative diagnosis of parasternal omental hernia is usually based on the radiographic demonstration of a density in either cardiophrenic angle and cephalad displacement of a portion of the transverse colon. We wish to emphasize the use of a pneumoperitoneum as an accessory diagnostic aid.

The differential diagnosis of masses at the cardiophrenic angle involves consideration of the following: lymphomas, teratomas, lipomas, neurofibromas, aberrant thyroids, celomic cysts, and peripheral lung neoplasms. Our recent experience with the following three cases demonstrates the application of various roentgen procedures and may serve to evaluate their merits.

Parasternal omental hernias occur relatively infrequently. In 1937, von Gryerz,[5] discovered in the literature six cases diagnosed during life by roentgenogram. Colmers,[1] in 1941, reviewed the literature and found 120 cases of various types of parasternal hernias. These included the six cases mentioned here. He found one case of parasternal omental hernia in which the diagnosis was suggested by chest roentgenogram and later proved by exploration. This case was reported by Meade and Ravdin.[10]

CASE REPORTS

CASE 1.—J. B. L., a white woman, aged 50 years, was admitted to Emory University Hospital in September, 1946. Chief complaints were fullness in the right side of the chest and a productive cough. The past history revealed a clinical diagnosis of pulmonary tuberculosis in 1918. Following hospitalization for two years, the patient was discharged with arrested disease. On a chest roentgenogram made in 1942, a small mass in the right lower lung field was discovered. This density subsequently increased in size. The physical findings referable to the chest consisted of dullness to percussion over the right lung base and absent breath sounds over the same area. Roentgenologic studies on admission showed the presence of a mass measuring about 9 cm. in diameter at the right cardiophrenic angle anteriorly (Fig. 1). A bronchogram disclosed separation of the right middle and lower lobe bronchi with partial obstruction of the secondary branches of the right middle lobe bronchus. A barium enema performed elsewhere revealed cephalad displacement of the transverse colon anteriorly with this portion of the colon having the appearance of an inverted "U" (Fig. 2). On bronchoscopy, separation of the right middle and lower lobe bronchi was observed.

An exploratory thoracotomy revealed a hernia of omentum through the right foramen of Morgagni with the sac presenting itself between the middle and lower lobes of the right lung. The hernia consisted of a peritoneal sac with partial incarceration of omentum.

CASE 2.—A. A., a white woman, aged 68 years, was admitted to Emory University Hospital in February, 1947, complaining of nervousness. One week previously, a routine roentgenogram of the chest showed the presence of a mass in the right cardiophrenic angle.

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Fig. 1.

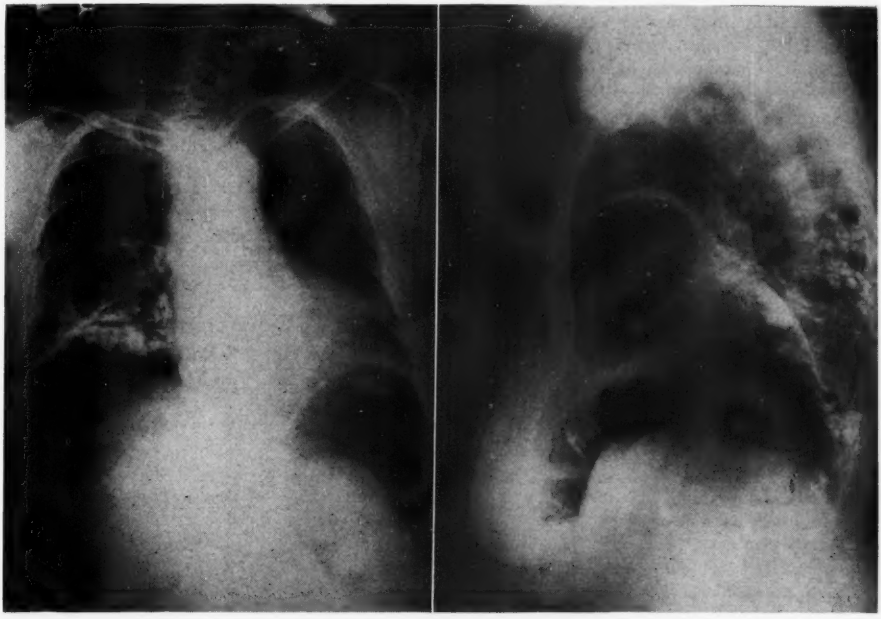
Fig. 2.

Fig. 1 (Case 1).—Mass in right cardiophrenic angle.
Fig. 2 (Case 1).—Cephalad displacement of transverse colon as a result of omental hernia.

Fig. 3.

Fig. 4.

Fig. 3 (Case 2).—Mass in right cardiophrenic angle.
Fig. 4 (Case 2).—Cephalad and anterior displacement of transverse colon due to parasternal omental hernia.



A. *B.*

Fig. 5, *A* and *B* (Case 2).—Posteroanterior and lateral roentgenograms of the chest following introduction of pneumoperitoneum. Note air in hernial sac.

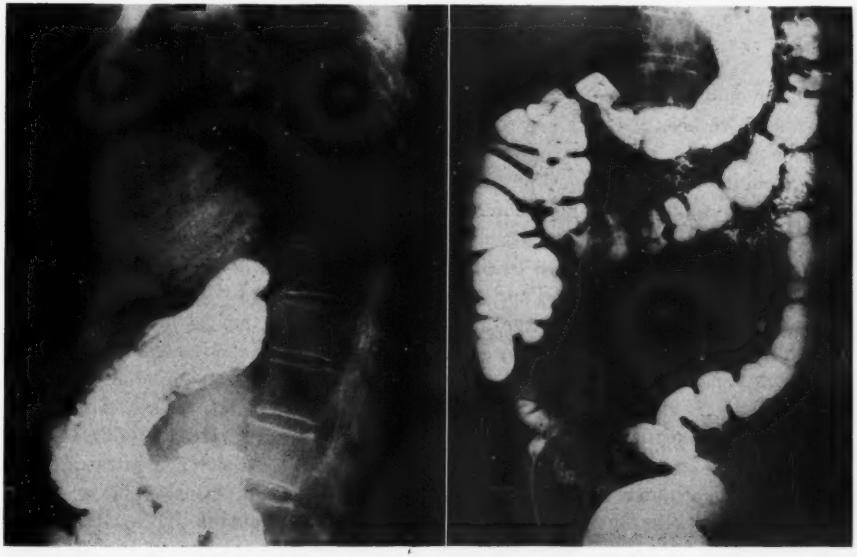


Fig. 6. Fig. 7.

Fig. 6 (Case 3).—Lateral roentgenogram of chest and abdomen. Note mass in anterior costophrenic sulcus.

Fig. 7 (Case 3).—Parasternal omental hernia. Colon and stomach in normal position.

Physical findings referable to the chest were occasional râles over the middle lobe of the right lung. Sputum studies and bronchoscopy revealed no additional information. Roentgenologic examination of the chest showed a large oval-shaped mass at the right cardiophrenic angle anteriorly (Fig. 3). A lateral film of the abdomen demonstrated a gas-filled loop of colon which was displaced cephalad and anteriorly beneath the dome of the right diaphragm. This finding was confirmed by a barium enema (Fig. 4). Following the introduction of a pneumoperitoneum, air was demonstrated within the right cardiophrenic angle soft tissue mass (Fig. 5, *A* and *B*). A tentative diagnosis of parasternal omental hernia was made from these findings, and the patient was discharged without surgery because of age and absence of symptoms.

CASE 3.—A. O., a white woman, aged 50 years, was admitted to Emory University Hospital in December, 1947, complaining of pain in the right side of the chest and a productive cough of three months' duration. Two months prior to admission, a roentgenogram of the chest showed an area of increased density at the right lung base compatible with the diagnosis of a virus pneumonitis. Additional roentgen study two weeks later showed no change in the appearance of the lung fields. On physical examination, definite râles were heard at the right lung base. Bronchoscopy, sputum, and blood studies revealed no additional information.

Roentgenologic examination of the chest showed a soft tissue mass at the right cardiophrenic angle anteriorly (Fig. 6). Compression of the secondary branches of the right lower lobe bronchus was observed from bronchography. On barium enema, the colon assumed a normal position (Fig. 7). X-ray pictures taken after pneumoperitoneum showed no air within the intrathoracic mass.

An exploratory thoracotomy revealed a hernia 5 by 8 cm. between the middle and lower lobes of the right lung, which consisted of omentum in a peritoneal sac. The neck of the sac was obliterated by dense adhesions. The mass presented through the foramen of Morgagni on the right.

The patient made an uneventful recovery and now has no symptoms referable to the chest.

DISCUSSION

The embryology, anatomy, etiology, and classification of parasternal omental hernias have been discussed in detail by Colmers[1] and others,[7, 3] and no attempt will be made here to enlarge upon their work.

Robbins[11] and Harrington[6, 7] have emphasized the importance of the change in position of the transverse colon in parasternal omental hernias. With barium enema, the transverse colon assumes an inverted "V" pattern with the apex pointing toward the foramen of Morgagni. In one of the cases the transverse colon appeared less angulated and assumed the contour of a "U."

It has been found that the abnormal position of the gas-filled transverse colon may be suspected on a lateral roentgenogram of the abdomen. This finding is shown to advantage in Fig. 8.

Sielman,[14] in 1924, demonstrated the presence of a hernial sac containing colon above the diaphragm utilizing pneumoperitoneum. He was able to differentiate the wall of the hernial sac from the wall of the large intestine. A small pneumothorax complicated this diagnostic procedure. Hayer,[8] in 1933, warned of the complications of diagnostic pneumoperitoneum. These consisted of peritoneal adhesions and gangrene of the stomach with perforation from kinking of gastric arteries. In our experience, no complications resulted from the use of this procedure.

Sante,[13] Carman,[2] and Faulkner[3] have all suggested the use of pneumoperitoneum in the diagnosis of subdiaphragmatic hernias. We have been unable to find in the American literature available to us a reference to the specific use of pneumoperitoneum in the diagnosis of congenital parasternal hernias containing omentum.

In Case 1, the abnormal position of the transverse colon was present, suggesting the correct diagnosis. In Case 2, the diagnosis was established without any doubt by pneumoperitoneum, although the characteristic sign of the barium enema might have sufficed.

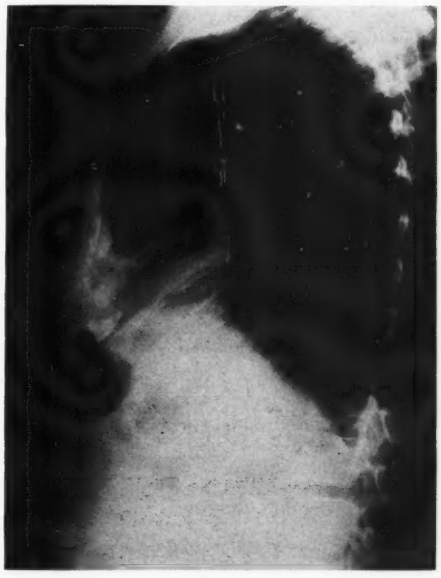


Fig. 8 (Case 1).—Lateral film of chest and abdomen showing the abnormal position of the gas-filled transverse colon.

Of considerable importance is Case 3. In this patient none of the roentgen criteria used in Cases 1 and 2 gave evidence of the correct diagnosis.

There may be cases in which, in spite of negative barium enema studies, a pneumoperitoneum will reveal positive information. It is believed that, in these cases, pneumoperitoneum will have its greatest usefulness.

SUMMARY AND CONCLUSIONS

1. Certain roentgenologic studies are invaluable aids in the diagnosis of parasternal omental hernias. It is essential that these benign lesions be differentiated from neoplasm.

2. The roentgenologic findings may be as follows: (a) a soft tissue density at either cardiophrenic angle; (b) the change in position of the gas-filled transverse colon on a conventional lateral film of the abdomen; (c) the cephalad dis-

placement of the barium-filled colon assuming the contour of an inverted "V" or "U"; (d) the presence of air within the soft tissue mass following pneumoperitoneum.

3. There are certain cases in which none of the characteristic roentgen findings are present, making a definite preoperative diagnosis impossible.

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AORTOESOPHAGOGASTRIC FISTULA, AN UNUSUAL COMPLICATION OF ESOPHAGOGASTROSTOMY PERFORMED UNDER THE AORTIC ARCH FOLLOWING ESOPHAGEAL RESECTION FOR CARCINOMA

A Report of Two Cases

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ESOPHAGEAL resection for carcinoma with the restoration of the continuity of the gastrointestinal canal now is a well-established procedure. The mortality of this procedure is related to (1) the older age of the patients afflicted with this disease, (2) the problem of anesthesia, (3) the location of the lesion with regard to its position above or below the aortic arch, (4) the technical difficulties associated with any anastomosis of the esophagus, and (5) the complications of infection within the thoracic cage.

It is apparent that certain of the complications which have resulted in a rather high mortality rate must be considered as unavoidable because of the age of the patients combined with a disadvantageous location of the lesion. At the present time the morbidity and mortality are increased markedly when the resection and anastomosis must be performed above the arch of the aorta. Undoubtedly, as more experience is gained with the higher lesions, a better knowledge of the most satisfactory method will be the result.

The complications which must be considered as being avoidable are related mainly to the problems of technical difficulties, anesthesia, and infection, either primary or secondary to trauma and/or leakage in the area of the anastomotic line. Although the antibiotics have altered the prognosis of infection in general, the problem of its avoidance in the chest still is of paramount importance.

The recent observation of two cases exhibiting an unusual complication following esophagogastrostomy has prompted this presentation. No previous report has been found describing a similar complication. In both instances, the patient died of an exsanguinating gastrointestinal hemorrhage from an aortoesophagogastric fistula.

CASE REPORTS

Case 1 (No. 763980).—R. S., a 63-year-old man, was admitted to the University Hospital on April 9, 1946, and died on May 4, 1946.

History.—The patient gave a history of dysphagia, regurgitation, and epigastric distress for the previous three months. There had been a twenty-pound weight loss in the past month. Immediately prior to admission, he was able to take only liquids augmented by some soft food. There was a history of two previous cerebral accidents having occurred thirteen and seven years, respectively, prior to admission.

Physical Examination.—Examination revealed a somewhat asthenic, deaf, white man with a right hemianopsia and a right hemiplegia. The blood pressure was 182/100, respirations were 22, and pulse was 80.

Laboratory Findings.—The hemoglobin, white blood cells, urine, Kline and Kahn, blood urea nitrogen, plasma protein, venous pressure, and vital capacity were either negative or within normal limits.

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On March 21, 1946, a gastrointestinal study revealed a carcinoma of the lower mid-portion of the esophagus.

A roentgenogram of the chest revealed a nodular density in the left base, possibly representing a metastatic nodule. There was a small area of pneumonia in the right middle field.

The electrocardiogram was thought to be abnormal. No definite diagnosis was suggested on the basis of this examination.

The patient was prepared for surgery by the oral route for a period of thirteen days by means of a liquid high protein, high carbohydrate, high caloric diet.

Operation.—On April 22, 1946, the patient was taken to the operating room for an exploratory thoracotomy on the left side, through the bed of the fifth rib, which was removed.* The carcinoma was noted to begin approximately 2 or 2½ cm. below the inferior border of the arch of the aorta. This had perforated into the adjacent tissues. It was felt that there were probably metastases in the hilus of the left lung and some metastatic nodules along the superior border of the pancreas in the abdomen. The lesion was entered into in the course of dissection, and the operator was committed to excision of the lesion. An esophagogastrostomy was performed just at the inferior border of the arch of the aorta without undue tension. Interrupted silk sutures were used. The diaphragm was sutured about the stomach.

Pathologic Report.—

Gross: The specimen consisted of a segment of esophagus measuring 8 by 5 cm. The mucosal aspect presented an ulcerated nodular elevation. The edge of the lesion extended to within 3 mm. of the proximal line of resection.

Microscopic: The tumor was composed of poorly differentiated, squamous epithelial tumor cells growing in irregular masses and clumps; in the center of some, necrosis was present, and others showed only cornification. The proximal and distal lines of resection were free of tumor. The tumor had penetrated all layers of the esophagus and was growing on the surface of the specimen. No lymph nodes could be found.

The conclusion was a squamous-cell carcinoma of the esophagus.

Postoperative Care.—The patient's temperature varied from 99 to 103.2° F. with a corresponding increase in pulse rate without any remarkable increase in the respiratory rate. A roentgenogram on April 29, 1946, revealed a small residual pleural effusion in the left base. On May 2, 1946, the chest examination revealed minimal infiltration on the upper lobe on the right. On physical examination on this day, a pleural friction rub was heard over the chest anteriorly. At 6:55 A.M. May 4, 1946, the patient experienced a sudden onset of dyspnea, cyanosis, pain in the chest, and apprehension. Almost immediately he had a small "hemoptysis." A subsequent "hemoptysis" occurred at 8:15 A.M. Some pulmonary edema was present. The blood pressure was 100 systolic, and the examiner was unable to determine the diastolic pressure; the pulse was 120. The impression at that time was large pulmonary infarct, although there were no signs of thrombophlebitis or phlebothrombosis of the lower extremities. At 8:30 A.M. a bilateral cervical sympathetic block was performed with 1 per cent novocain. The patient died approximately fifteen minutes later.

Autopsy Examination.—At the point of anastomoses there was a fistula between the aorta and esophagogastric anastomosis apparently created by a suture which passed through the media of the aorta and the esophagogastric anastomosis. This had resulted in a large hemorrhage from the aorta through this fistula into the stomach and bowel. This blood extended up behind the esophagus into the neck, behind the great vessels and along the paravertebral spaces. The remainder of the gastrointestinal tract contained approximately 3,000 c.c. of fresh blood. This figure of 3,000 represented the measured amount of blood. In addition, there was a large volume of blood which poured out of the bowel when it was opened. Examination of the pancreas revealed it to be hard and indurated along the superior border where it was matted to several celiac nodes.

The examination of the brain revealed: (1) cerebellar arteriosclerosis and (2) old encephalomalacia.

The conclusions were: (1) metastatic squamous-cell carcinoma of the celiac nodes with extension into the pancreas; (2) an aortoesophagogastric fistula with an extensive hemorrhage from the aorta through the anastomotic line into the gastrointestinal tract and into the mediastinum.

*Operation by a colleague, assisted by K. A. M.

CASE 2 (L-1663).—R. L., a 50-year-old white man, was first admitted to the St. Johns Hospital, St. Paul, Minn., and was transferred to the University Hospital on Nov. 24, 1947, and discharged on Jan. 5, 1948.

History.—The patient gave a history of anorexia, some pain in the epigastrium, and minimal dysphagia of approximately six weeks duration. The patient had lost twenty-five pounds in this period.

Physical Examination.—The physical examination disclosed a somewhat emaciated adult man. The remainder of the examination was essentially negative.

Laboratory Findings.—The laboratory findings revealed a normal blood picture and urine. On an x-ray examination of the chest, the heart and lungs were found to be normal. A gastrointestinal study was performed, at which time a transient obstruction to the flow of barium in the esophagus at the level of the ninth and tenth dorsal vertebra was observed. There appeared to be a slight constriction of the esophagus in this area and a small mass on the posterior wall projecting into the lumen of the esophagus. The esophagoscopist reported a small granulating area at the point of the junction between the middle and lower third of the esophagus. A biopsy was taken.

The microscopic examination of this area showed infiltrating aberrantly growing masses of squamous epithelium.

The diagnosis was a squamous-cell carcinoma of the esophagus.

Operation.—The patient was prepared for surgery, and on Dec. 9, 1947, a left posterior transthoracic approach excising the sixth rib was performed (K. A. M. assisted by E. C. E.). No palpable nodes were present. The lesion in the esophagus was found at the junction of the middle and lower third of the esophagus and was the size and shape of a small lemon. The esophagus proximally was sacrificed immediately distal to and underneath the arch of the aorta. The distal line of resection was through the junction of the proximal one-fourth and distal three-fourths of the stomach. The left gastric artery was amputated at the site of its exit from the celiac axis. The distal stomach then was closed by running sutures supplemented by an additional row of 0000 Deknatel sutures. The spleen was sacrificed during the operative procedure. An esophagogastrostomy was performed in the usual fashion. Two catheters were placed in the chest, one posteriorly along the posterolateral gutter, and the other was placed at the site of the anastomosis. Care was taken to wrap the anastomosis with a substantial portion of the gastrosplenic ligament. This was not sutured in place.

Postoperative Course.—The early postoperative course was smooth; the highest elevation was 100.2° F. on the fourth postoperative day. The temperature then fell to normal the following day. During the early postoperative days difficulty was encountered in the expansion of the left lung. On the ninth postoperative day the temperature, having been normal for five days, rose to 102° F. By means of aspiration and the insertion of additional catheters in the chest under water-seal drainage, the lung finally re-expanded. On the seventeenth postoperative day a foul drainage was noted from the intercostal catheters. The presence of a fistula was suspected. Methylene blue given by mouth confirmed the diagnosis. The patient who had been eating six small feedings daily was taken off all feedings by mouth with the exception of one ounce every hour of distilled water containing 1,000 units of penicillin per cubic centimeter. Within three days the fistulous tract could be no longer demonstrated. The empyema cavity then was irrigated with Dakin's solution every four hours. The remainder of the convalescence was uneventful, and on Dec. 30, 1947, after the instillation of 10 c.c. Lipiodol, an open thoracotomy was established through the bed of the ninth interspace posteriorly. The patient was discharged six days after the last operative procedure.

Follow-up.—Within a short period of time the empyema cavity had healed and the thoracotomy tube was removed. The patient was able to take semisolid food without difficulty in swallowing. An x-ray examination on Jan. 14, 1948, revealed some slight area of narrowing at the point of anastomosis. There was evidence of a blind tract extending from the line of anastomosis laterally for approximately 1 cm. and approximately 3 or 4 mm. in diameter. Dilation was carried on with the Vincent type dilator. A No. 21 French dilator was passed without difficulty. Subsequently, a No. 31 French followed by a No. 37 French dilator was passed without undue difficulty.

On Feb. 27, 1948, following a rather large dinner, the patient complained to members of his family of a rather sudden severe substernal pain and dyspnea and shortly became apprehensive. This was followed by a moderate-sized hematemesis. He was admitted immediately to the hospital.

Second Hospital Admission.—The patient was admitted at 8:45 P.M. on Feb. 27, 1948, and died at 1:00 P.M. on Feb. 28, 1948.

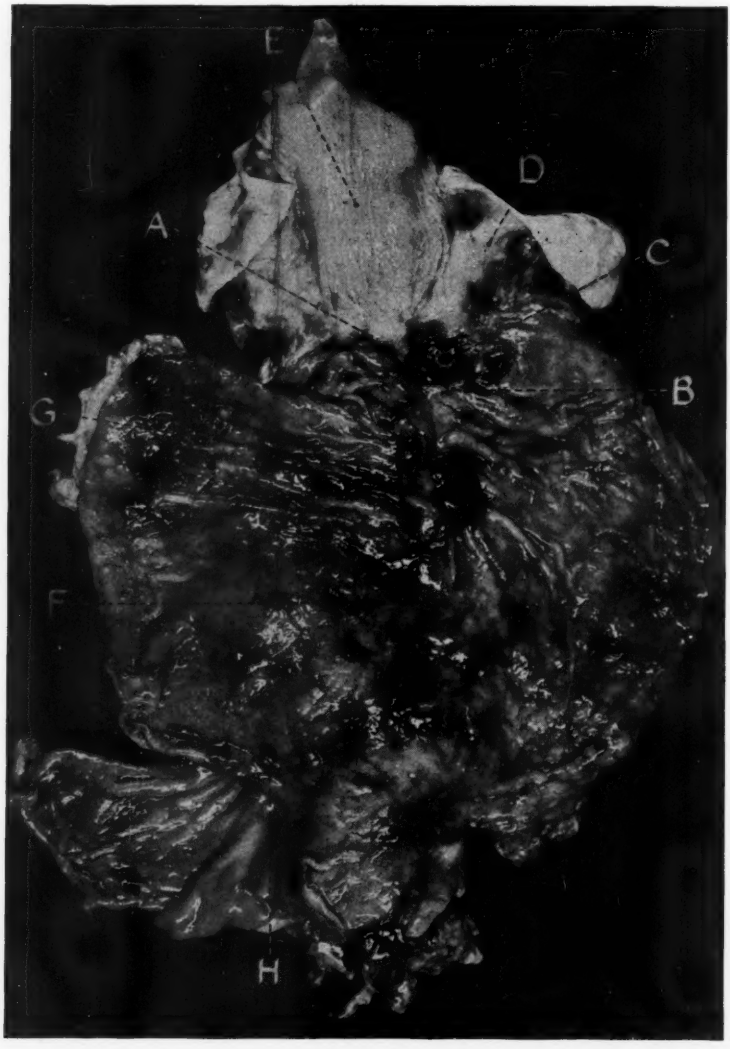

Fig. 1.—Autopsy specimen. The esophagus and stomach have been opened anteriorly in the midline. *A*, Anastomosis of the esophagus to the stomach; *B*, ulcer (probably represents a separation of the anastomosis) at the esophageal gastric anastomotic line; *C*, aorto-esophagogastric fistula; *D*, arch of the aorta; *E*, esophagus; *F*, residual stomach; *G*, line of inversion of the distal residual gastric pouch; *H*, pyloric sphincter.

The patient was entirely conscious upon admission. The blood pressure was 88/64, pulse 120, respirations were 24. Shock blocks were inserted under the foot of the bed, and morphine sulfate was administered. Periodically he continued to vomit varying amounts of bright red blood. (The largest measured amount was 1,300 c.c.) Although the patient appeared to be desperately ill, he continued to be conscious and responded well to questioning up until his death the following day at 1:00 P.M. The total amount transfused before death was 5,000 c.c. of blood, calcium having been given during the night.

Because of the previous experience of observing an aortoesophagogastric fistula following an esophagogastrostomy in this location, this diagnosis was entertained in this

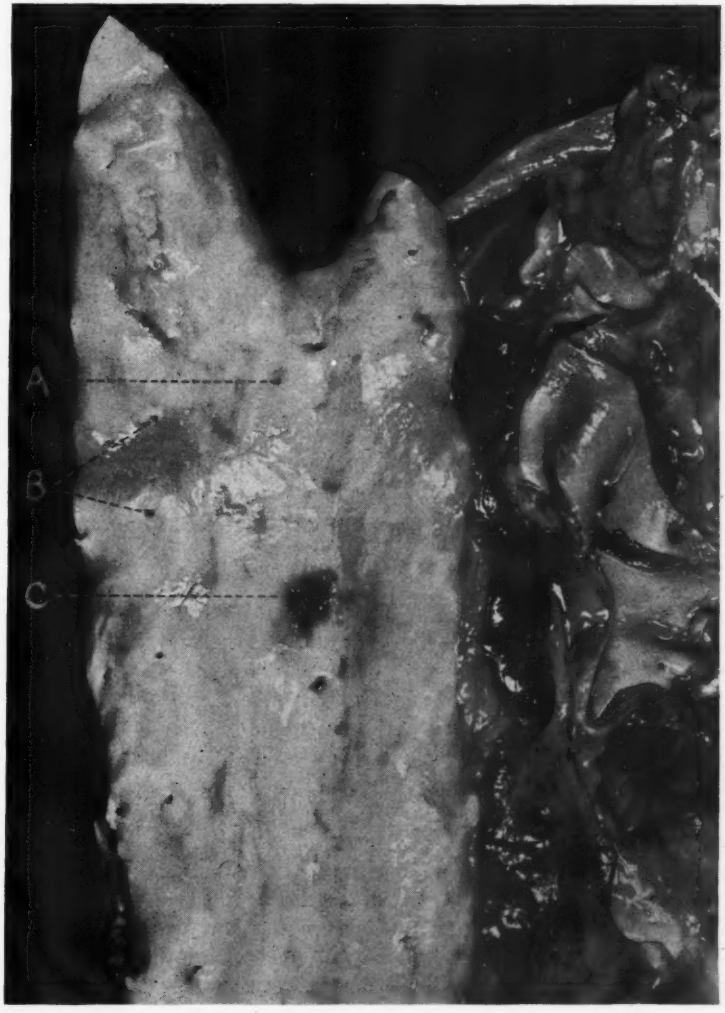

Fig. 2.—Autopsy specimen. The aorta has been opened posteriorly in the midline. *A*, Arch of the aorta; *B*, orifices of the intercostal vessels; *C*, aortoesophagogastric fistula.

patient. At no time during the patient's second admission to the hospital was he in a condition sufficiently satisfactory for an exploration of the site of the anastomosis to be contemplated.

Autopsy Examination.—At autopsy* an aortoesophagogastric fistula was found which measured 6 mm. in diameter on the aortic side. Within the lumen of the esophagus and stomach corresponding in position with the aortic perforation, an ulcer (2 cm. in diameter) at the esophagogastric anastomosis on the left lateral side was found (see Figs. 1 and 2). There was no evidence of recurrent tumor. An incidental finding was the presence of a small area of irregular glands in the prostate which were histologically malignant. Microscopically, the ulcer contained acute and chronic inflammatory cells. The margin of the opening in the aorta was necrotic and also was infiltrated with inflammatory cells.

DISCUSSION

The proximity of the aorta to the line of anastomosis undoubtedly carries with it an additional hazard. If trauma to the aorta is avoided and primary healing of anastomosis occurs, this complication probably will not occur. In each of the cases presented, one of these factors played a role in the development of the aortoesophagogastric fistula.

In the first case, the aorta was traumatized by a misplaced suture passing through the media of the aorta and the esophagogastric anastomosis. In the second case, in spite of a carefully performed completely satisfactory anastomosis, leakage occurred at the suture line (seventeenth postoperative day) with the development of an esophagogastric pleural fistula. This area almost immediately was sealed by the juxtapositioned aorta. It is conceivable that such a fistula might communicate with the main pulmonary artery, the inferior pulmonary vein, or the bronchial vessels. The major pulmonary vein is well protected by the interposed bronchus. The vessel involved, of course, would depend upon the level of the anastomosis. However, the danger of a venous erosion is less likely. It is well known that in the presence of infection because of the constant pulsation of an arterial vessel, perforation is more likely to occur in an artery than in a vein similarly exposed.

It is apparent that in order to protect the patient against the possibility of such a complication, it is important to avoid direct contact of the anastomotic line with any major vessel in the area. This can be accomplished readily by the interposition of a satisfactory tissue between these structures. In the second case, with the observation of the first catastrophe still fresh in mind, a large portion of the gastrosplenic ligament was spared in the abdominal dissection for the express purpose of wrapping it about the anastomosis. This was done. At the time of the autopsy the aorta was adhered to the esophagogastric anastomosis and, in addition, the wall of the aorta formed the base of the ulcer found at the anastomotic level. This finding suggests that either a dissolution of the gastrosplenic ligament took place or the position of this tissue was altered when the patient assumed the upright position on the first postoperative day. In retrospect, the choice of tissue was a poor one. The gastrosplenic ligament receives the major portion of its blood supply from the vasa brevia derived from the distal portion of the splenic artery. These arterial vessels usually are divided

*By Dr. John F. Noble.

when a concomitant splenectomy is performed and/or sacrificed when the stomach is mobilized for its craniad intrathoracic displacement. In order to obtain a tissue with an intact blood supply, a portion of the gastrocolic ligament which is well nourished by the right gastroepiploic artery should be utilized. Furthermore, in order to be assured that this tissue will remain in position when so placed, it should be sutured carefully about the esophagus and the retroesophageal tissue. In this fashion any direct contact between major vessels in this area and the esophagus is obviated.

There are certain common features in these cases which should allow serious consideration of a diagnosis of aortoesophagogastric fistula. In each instance an esophagogastrostomy had been performed immediately distal to and under the arch of the aorta. This fact, combined with the subjective findings of sudden pain in the chest accompanied by dyspnea and apprehension, plus the objective findings of a varying degree of shock accompanied by hematemesis, should allow the presumptive diagnosis to be made. In the second case, there was no vomiting of blood in the usual sense but rather a regurgitation of the blood without wretching or any obvious effort.

Granted that the patient's condition can be improved sufficiently by massive transfusions to allow the consideration of a surgical approach, the arterial defect could be explored and undoubtedly, in certain instances, closed without great difficulty. Certainly there is everything to gain and nothing to lose by such a maneuver. In Case 2, although the tissue about the perforation was necrotic for approximately 1 mm. with acute and chronic inflammatory cells present, the consistency of the aorta 1 or 2 mm. peripheralward about the circumference of the fistula was of sufficiently good quality to allow a satisfactory closure of the defect.

Only by such an aggressive approach can the life of a patient who otherwise is doomed be salvaged.

SUMMARY

The unusual complication of an aortoesophagogastric fistula resulted in the death of two patients herein reported. Both patients had been subjected to esophageal resections for carcinoma with the restoration of the continuity of the gastrointestinal tract by an esophagogastrostomy distal to and underneath the arch of the aorta. Certain aspects concerning the avoidance of this complication have been discussed. The clinical features observed in these two cases have been presented. The consideration of this complication as a diagnostic possibility has been stressed. The feasibility of a surgical closure of the defect has been predicted.

ANEURYSM OF THE FIRST PART OF THE LEFT SUBCLAVIAN ARTERY

Review of the Literature and a Case History

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EVER since the second century, when Antyllus (works of Oribasius) first described what is now the modern surgical treatment of aneurysms, ligature of the first part of the left subclavian artery must have been in the nature of a challenge to surgeons. No one can fail to be thrilled by Halsted's words (1920):

> In ligating the first portion of the left subclavian in the chest the operator may not, as formerly, be more greatly impressed by the magnitude and cleverness of his performance than by the miraculous effect of the ligation of the artery upon the great pulsating tumour which with each beat of the heart jarred the whole frame of the sufferer. The moment of tying the ligature is indeed a dramatic one. The monstrous booming tumour is stilled by a tiny thread, the tempest silenced by the magic wand.

It is true that many men revered in surgery have condemned the operation as impossible. On reading the accounts of earlier operations, particularly those of the pre-Listerian era, this attitude can be understood. Of the 21 cases collected by Halsted up to 1920, there were 8 patients who died and only 8 were known to be cured. In only 2 patients did the aneurysm originate as far medially as the junction of the first and second parts of the artery. In all except 1 the ligation was undertaken from above and anteriorly by dividing the sternomastoid after resecting a portion of the clavicle and sometimes resecting the manubrium and even one or two inches of the first rib. Even in the patients who recovered, the morbidity was high, due to damage to the thoracic duct or the pleura or sepsis resulting on tamponade for uncontrollable bleeding. Halsted's critical comments on each of the cases described are still invaluable, but although when he wrote his monograph Morriston Davies, Sauerbruch, and others had shown that the pleural frontier could be violated with impunity, the idea of a transpleural ligation does not seem to have occurred to him. In only one of the cases he describes was a different approach used. Sherrill (1911), operating for what he believed to be a subclavian aneurysm, resected three inches each of the second, third, and fourth ribs posteriorly and approached the mediastinum extrapleurally. He found it easy to ligate the subclavian, but unfortunately the aneurysm originated in the aortic arch.

Lee, Mitchell, and Peacock (1934) described a successful operation by the superior approach for a traumatic aneurysm of the first part of the left subclavian artery. From the literature they were able to collect 128 cases of traumatic aneurysm of a subclavian artery since 1767, including Halsted's and

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Souchon's (1895) collected cases. The only additional cases, however, in which quite indisputably the first part of the left subclavian was ligated, were those of Von Haberer and their own.

Von Haberer (1916) operated on 72 patients with aneurysm due to gunshot wounds, 7 of which were in the left subclavian artery. Four of these involved the first part of the artery and were exposed by division of the clavicle. In 2 patients, after excision, an anastomosis of the arterial ends was performed. There was one death in this series. The remaining 3 patients were dealt with by supraclavicular incisions, with an anastomosis in one.

Kirchner's (1927) case was due to a wound received in 1917, and the patient was operated upon in 1927 by a supraclavicular route.

A search of the literature since then reveals comparatively few cases, except those associated with cervical ribs. These, I feel, come into a separate category, partly because the aneurysm always forms distal to the point of compression (MacFee, 1940) and is thus readily accessible to the surgeon, and also because the condition is almost always relieved by removal of the offending rib. Lindskog and Howes (1937), in addition to reporting a case, reviewed the literature of aneurysms associated with cervical rib and reached similar conclusions.

An important step forward was made by Henry (1923) who, from the dissecting room, described a posterior mediastinal approach to the first part of the left subclavian. The soft tissues are turned up as a medially hinged flap, and the back end of the second rib and the transverse process are resected. The pleura is stripped from the mediastinum and the origin of the artery exposed. He quoted Sherrill's case as the only patient actually operated upon by posterior mediastinotomy.

Wright Smith (1927) reported a patient with syphilitic aneurysm of the second and third parts of the left subclavian operated upon by Hailes. Hailes resected the posterior ends of the second and third ribs to reach and ligate the first part of the artery. Thus, although Wright Smith called it a Henry operation, it was much more like Sherrill's original approach.

Nelson (1932) quoted a case by Alexander, who approached a right subclavian aneurysm by a similar route, but on this side it was less successful, and after a subsequent operation from the front to ligature the innominate artery, the patient died. Incidentally, during the posterior mediastinotomy, the pleura was unintentionally widely opened.

Henry's approach, however, found approval. Matas (1926), reviewing twenty-five years experience, mentioned six ligations of the first part of the left subclavian for aneurysm, only two being intrathoracic. The approach was always the classical one from above and in front, but he concluded by recommending Henry's operation. Similarly Elliott (1932), although he described no relevant case in his discussion on subclavian aneurysm, commended Henry's approach.

Muller (1935) described a traumatic aneurysm of the second and third part of the left subclavian. He ligated the first part of the artery by the anterior extrapleural route and subsequently successfully excised the tumor. He reviewed the literature and discussed the subject. Worth mentioning is his comment from

Elliott's earlier paper (1912), that for aneurysm of the third part of the subclavian the first part of the artery should be ligated as a preliminary. In view of the case now to be reported, two points in his paper are particularly significant. After careful stripping of the pleura in the anterior approach, it became torn, but no ill effects resulted. In his discussion he stated that if the aneurysm was in the first part of the left subclavian no treatment was possible except distal ligation.

The literature of World War II does not seem to include many relevant cases. Adams (1946) reported six subclavian aneurysms but did not give details. In each case the approach was through or above the clavicle.

Schumacker and Carter (1946), reviewing arteriovenous fistulas and arteriovenous aneurysms in military personnel, described 364 cases. These included 10 subclavian fistulas and 5 aneurysms with 1 operative death in the latter group. Side and site of the lesions were not stated nor was the operative approach described.

Elkin (1946) discussed 450 cases of aneurysms and arteriovenous fistulas in military personnel. In 45 patients part of the clavicle was resected but these included carotid, innominate, subclavian, and axillary aneurysms, with proportions not stated. Significantly, the only death was in a patient in whom the first part of the left subclavian was affected, and was due to failure to get a suture on the proximal end of the artery through the exposure used.

An anterior combined thoracic and cervical approach to the left subclavian was described by Wilson and Carr (1948). In a case of arteriovenous aneurysm they divided the sternomastoid, removed the medial three-quarters of the clavicle, divided all the ribbon muscles, and split the sternum to the level of the third rib and cut across laterally to aid retraction. They then proceeded with an extrapleural exposure and successful treatment of the fistula.

Freeman (1946), describing 23 cases of aneurysm and fistulas due to war wounds, included an arteriovenous fistula of the left subclavian and the left innominate vein. The operative approach included division of the sternomastoid and strap muscles, resection of one-half the clavicle, and splitting of the sternum to the second cartilage. A successful repair was effected, but some wound infection necessitated reopening of the wound. The final outcome was satisfactory.

Rehn's cases (1942) appeared to concern the third part of the artery only. Other authors with extensive experience of war wounds of the chest supplied no relevant cases. These include Miscall and Harrison (1947) who reported 300 chest wounds, Martin (1947) who did follow-up on 106 cases for three months, Snyder and Tropea (1947) who reported 678 cases, and Tudor Edwards (1943) who, in his survey of war wounds and injuries to the chest, did not mention this particular injury. It is concluded that wounds involving the first part of the left subclavian resulted in death and not aneurysm.

For the sake of completeness it should be added that two articles which have been published in the Russian journals and two others by Bsteh (1947) and Perrotti (1947) could not be obtained, and it is not known whether or not the subclavian aneurysms which they described are relevant.

A search of the records of the Liverpool and North West Regional Chest Surgical Unit since 1940 revealed only one case of aneurysm of the first part of the left subclavian artery. This patient, a soldier 28 years of age, who sustained a gunshot wound of the left axilla at Anzio in February, 1944, was operated upon by Mr. H. Morriston Davies in June, 1944. The foreign body responsible for the aneurysm was accurately localized by roentgenogram and the chest opened by an incision into the third interspace from in front with division of the cartilages. The pleura was opened and the adherent lung freed. A nonpulsating swelling was found at the junction of the left subclavian and aorta corresponding to the site of the metallic foreign body. A needle was inserted into the swelling but only fibrinous fluid was obtained. A second needle was then inserted and blood under pressure forced up the piston of the syringe. At the same time blood began to spurt from the first needle hole. Both holes were oversewn and the chest closed. The patient suffered no ill effects but a roentgenogram one month later showed an increase in the size of the aneurysm. He was transferred to a Ministry of Pensions hospital and no further records are available. This case has been quoted in full, as it is the only one I have found recorded in which a direct intrapleural approach to the left subclavian artery has been made.

The report of my own case follows.

The patient was an engine driver, aged 40 years, who was admitted to the Liverpool Chest Surgical Centre on Aug. 3, 1948.

The history given was that he became ill with pneumonia of the left lung on May 8, 1948, and spent six weeks in bed. At the end of that time he was quite well, without cough or dyspnea, or pain in the chest. His own doctor examined him before sending him back to work and found abnormal physical signs, as a result of which roentgenograms were made and eventually the patient was sent to the Chest Unit. He had no complaints whatsoever at the time and was anxious to get back to work.

On examination he was a spare but healthy looking man, rather young for his age. The most striking feature was a fully developed left Horner's syndrome (Fig. 1). After questioning about this, he stated that in February, 1946, he was knocked down by a railway engine and fell striking his chest on a projection on the ground. He spent ten days in the hospital after this accident, but roentgenograms were not made; on discharge he noted a difference in the size of his eyes for the first time. The condition had never worried him greatly and had not altered in appearance since. There was no sign of finger clubbing in either hand, but the left hand was definitely warmer than the right. Radial pulses were equal.

On examination the upper left part of the chest, back and front, was stony dull to percussion, and breath sounds were absent. No other abnormality whatsoever was noticed. A roentgenogram of the chest showed a large rounded tumor extending from the apex of the thoracic cavity down to the seventh rib posteriorly and reaching medially to the mediastinum. This opacity was clearly defined and rounded. The rest of the lung fields were clear. (See roentgenograms illustrated in Figs. 2 and 3.) The tumor did not pulsate on screening.

Blood count was within normal limits. Wassermann reaction and Meinicke were negative, and a Casoni test proved to be negative. Sputum was mucoid and contained buccal flora only.

I had not much doubt that this was a case of neurinoma of some type, either originating from one of the intercostal nerves or, in view of the sympathetic paralysis of the eye and left upper limb, originating from the sympathetic chain as a ganglioneuroma. If the former, there was of course the possibility of an extension into the spinal canal; therefore, I asked

Mr. R. H. Hannah of the Liverpool Neuro-Surgical Unit to see the patient, and he agreed that there was no direct involvement of the central nervous system and he was prepared to be present at the operation and undertake the removal of any part of the tumor that entered the spinal canal.

Fig. 1.—Photograph of the patient showing the ptosis enophthalmos and small pupil, left side.

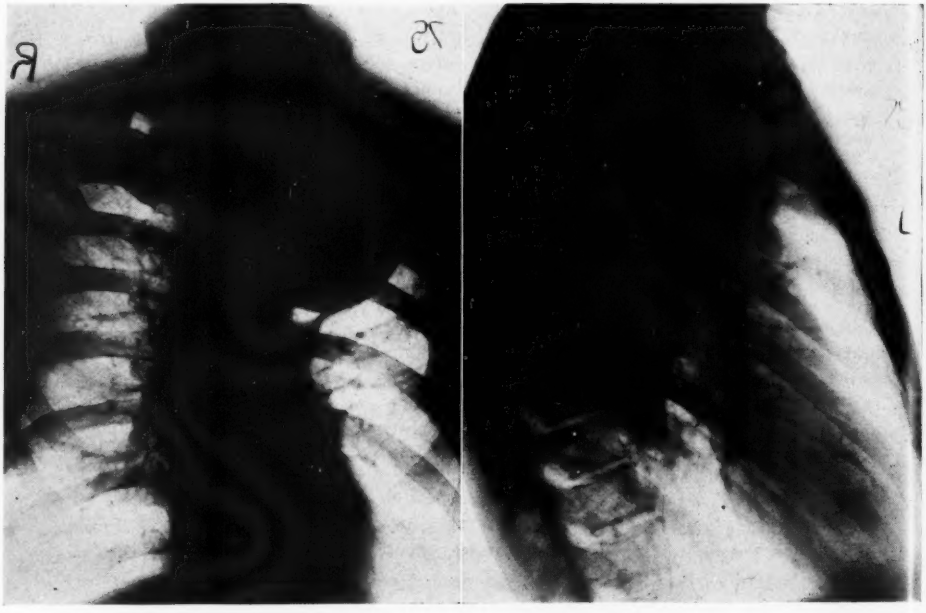

Fig. 2. Fig. 3.

Fig. 2.—Posteroanterior roentgenogram of the chest.
Fig. 3.—Lateral roentgenogram of the chest.

Operation was undertaken on Aug. 25, 1948. Anesthesia consisted of Tubarine, Pentothal, and nitrous oxide*; a blood transfusion was started running slowly in the saphenous vein before the operation was begun. The patient being in the lateral position, the fifth rib was resected and the fourth and third ribs divided posteriorly. The pleura was opened, but the pleural space was almost completely obliterated by adhesions, and the lung was separated only with considerable difficulty. A large tumor in the apex of the chest was closely adherent to the lung itself and could be separated only by sharp dissection, damaging the lung somewhat in the process. The tumor was further defined once the lung was freed and on the posterior surface stripped extrapleurally. It was found to be attached to the first thoracic nerve at its origin and was hard, not pulsatile, but somewhat cystic to feel. On medial dissection the subclavian artery appeared to run into the substance of the tumor within one inch of its origin from the aortic arch and only left it at the inner edge of the first rib. It was still not certain whether this was adherence or whether the tumor was an aneurysm (Fig. 4).

Fig. 4.—The tumor as seen at operation.

A purse-string suture was placed around one point of the aneurysm and a needle inserted. One hundred cubic centimeters of brownish altered blood were aspirated, after which the needle was withdrawn and the purse string tied. This produced some diminution in the tension of the cyst for the time being, but it soon became as tense as before, and during the later dissection, the purse string opened and this time bright red blood spouted from the needle puncture. Before proceeding further a loose thread was put around the origin of the subclavian and one at the inner border of the first rib. At this stage the subclavian was digitally compressed against the first rib, and the appearance of his left hand was noted. It was somewhat cyanosed, but the skin circulation seemed to persist. No radial pulse was palpable. It now seemed clear that the tumor was an aneurysm, and an attempt was made to separate it from the subclavian artery and narrow its pedicle. A

*Administered by Miss Noel Fenton.

large number of small vessels were dealt with and hemorrhage controlled, until on further separation of the aneurysm from the wall of the artery a very copious hemorrhage ensued. Traction on the two ligatures that had been passed around the subclavian failed to control it, so rubber-covered clamps were applied to the artery at the same points. The bleeding still occurred as soon as the compressing finger was removed. Later I found that one of the rubber-covered clamps was defective and it seems this was the cause of the failure to control hemorrhage by this method. Clamps were then applied directly to the bleeding part of the artery, the aneurysm was separated from it and dissected away leaving a considerable gap in the side of the subclavian artery. The mass stripped quite easily off the first thoracic nerve to which it was attached only by fibrous tissue. The subclavian artery was ligated and then transfixed one-half inch from its origin on the aortic arch and again immediately below the origin of the internal mammary artery. The intervening portion of the damaged artery was removed. The chest was closed with an underwater-seal drain. Examination of the sac showed it to be almost filled with laminated blood clot, and it was reported on by the pathologist* as follows: A cyst about 4 inches in diameter was received containing blood. The thin part of the wall consisted of compressed acellular connective tissue. A small thick portion showed organized clot into which new vessels were growing. The wall of this part was of chronically inflamed fibrous tissue, in which were identified blood vessels, nerves, and some islands of elastic tissue. The appearances were consistent with the diagnosis of aneurysm.

At the conclusion of the operation no left radial pulse was palpable, and the blood pressure could not be measured in the left arm. The hand was somewhat cyanosed, but the skin circulation was reasonably brisk. Nursing care included keeping the body as warm as possible, with the arm lying horizontally and at rest outside the bedclothes. At no time did he give rise to any anxiety whatsoever, and we could not stop him using the arm for lighting cigarettes, etc., after the first postoperative day.

Three weeks after operation he continued to have no left radial pulse, but there were no sensory changes and no loss of power in the arm. The cyanosis had disappeared and the hand remained warmer than the other one. The patient was then given permission to return to his job, which he was very axious to do. Most surprising of all, there had been a definite diminution in the ptosis of the left eye, although the pupil remained small.

DISCUSSION

Discussion centers on two aspects of the case, diagnosis and operative treatment.

Preoperative diagnosis was missed largely because insufficient attention was paid to the history. The history is given as it was received, and there was a tendency to dissociate the accident from the appearance of the Horner's syndrome, assuming that the stay in the hospital merely drew the patient's attention to a pre-existent state. Had the history been presented simply as that of an accident followed immediately by the appearance of Horner's syndrome and thereafter the discovery of an intrathoracic tumor, it is more likely that the diagnosis of aneurysm would have been reached. The failure to detect pulsation on screening is not surprising. The aneurysm in Mr. H. Morriston Davies' case did not pulsate, and Kerly (1939) stated that only 25 per cent of intrathoracic aneurysms show expansile pulsation on screening. The fact that there was no palpable tumor or pulsation in the neck is more significant. None of the cases in the literature reviewed was so completely intrathoracic as this one.

That the trauma of the patient's accident was sufficient to cause a partial rupture of the artery there is no doubt. While most of the traumatic cases described were due to penetrating wounds from shot or steel, some were due to

*Dr. Taylor.

simple fractures and not a few to comparatively minor injuries such as a fall on the football field. Simpson (1940-1941) described a number of fatal injuries to the intrathoracic vessels from minor trauma. Nelson, recording his case of innominate aneurysm, mentioned four other cases of aneurysm due to fracture of the clavicle, although no case involved the first part of the left subclavian artery.

During the operation, the question of anastomosis of the arterial ends did arise. In view of the fact that after occlusion of the artery an adequate blood supply to the limb was maintained, it was decided not to take the extra risks of an anastomosis. Although the word "adventurous" is perhaps no longer relevant, one may still ponder on Matas' words, "When operating, the surgeon should not obstinately insist upon re-establishing the continuity of the damaged artery by juggling with complicated, hazardous, uncertain and adventurous methods."

As I have pointed out, apart from Morriston Davies' unpublished case, I know of no direct transpleural approach to the left subclavian artery. In reading the accounts of the operations by other routes, one is impressed by the courage of the various surgeons who undertook them.

By contrast, the operation here described was a relatively simple one. An inspection of the interior of the left thorax reveals at once the whole of the first part of the subclavian artery and its internal mammary and costocervical branches before any dissection is undertaken. Surely this route, alone or combined with a supraclavicular incision in cases where the second and third part of the artery is also involved, is the method of choice. Injury to the pleura is frequently mentioned as a complication of both the anterior and posterior extrapleural approaches. Sometimes it produced undesirable results, sometimes it did not. If the opening is missed or is incompletely sutured, if the anesthetist has not adequate control of the lungs, if air remaining or fluid appearing in the pleural space be not evacuated, then indeed, opening of the pleura may be a disaster. If, however, a deliberate thoracotomy is undertaken, the complication rate will be negligible from this source.

It is undoubtedly presumptuous to argue on the strength of one case, but aneurysm of the first part of the left subclavian artery occurs only rarely in the practice of any surgeon. Diminution in the complications of this condition will occur only when it is recognized that the correct surgical approach to the artery, whether anteriorly or posterolaterally, is by direct thoracotomy.

It might not be out of place at this stage briefly to describe the collateral circulation likely to take place after ligature of the first part of the left subclavian artery (Fig. 5):

1. Between the vertebral artery and the vertebral artery of the opposite side and with muscular branches of the occipital artery
2. Between the inferior thyroid artery and the superior thyroid and other muscular branches from the carotid
3. Between the ascending branches of the transverse cervical and the deep cervical branch of the costocervical, anastomosing with descending branches of the occipital artery

4. Between the descending branch of the transverse cervical, the intercostal branches of the costocervical, the superior thoracic, the long thoracic, and pectoral branches of the thoracic axis anastomosing with the aortic intercostals
5. Between the internal mammary and inferior epigastric and aortic intercostals

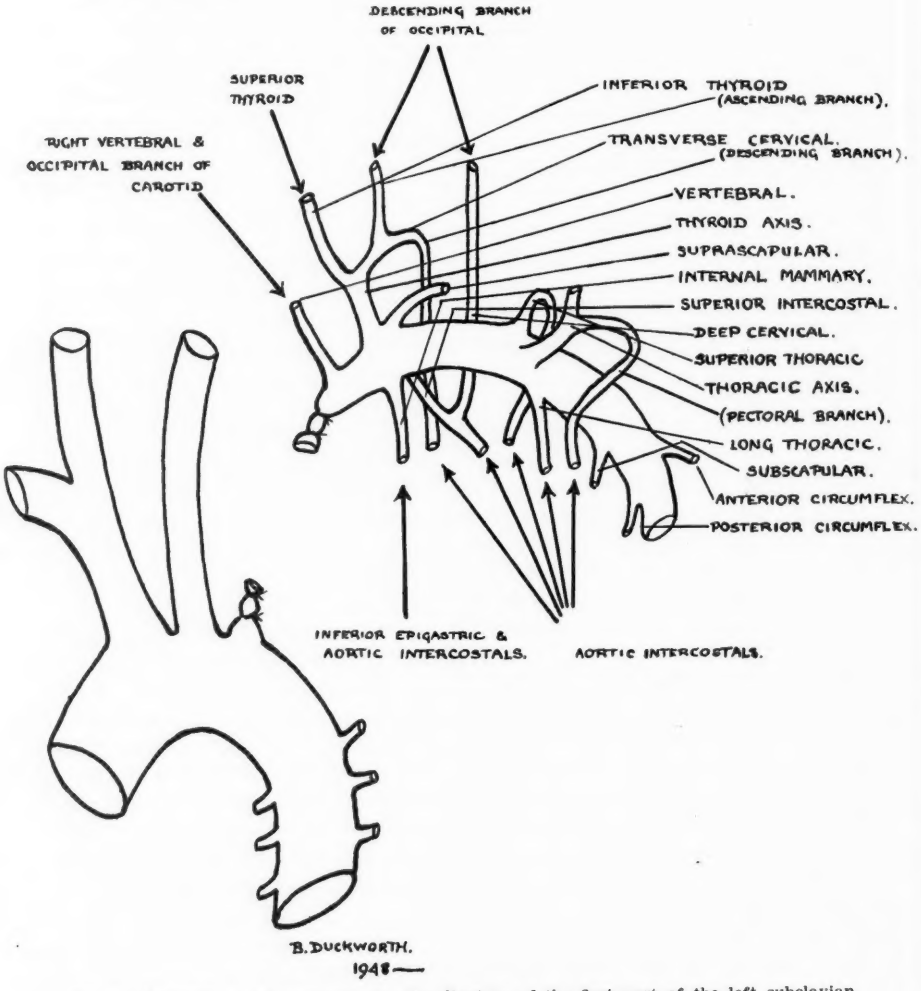

Fig. 5.—Diagram of the anastomosis after ligature of the first part of the left subclavian.

SUMMARY

The literature of aneurysm of the left subclavian artery and ligature of its first part is reviewed. An additional case is described and the diagnosis dis-

cussed. A plea for transpleural exposure of the first part of the left subclavian artery is made. The collateral circulation after ligature of the first part of the left subclavian is briefly mentioned.

In submitting this paper I most gratefully acknowledge my thanks to Miss Duckworth for the drawings, to Mr. H. Morriston Davies for his advice and encouragement, and to Mr. F. Ronald Edwards whose criticism and advice has been invaluable.

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*These articles are of particular importance.

THE TREATMENT OF PULMONARY HYDATID DISEASE

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IN A most interesting and instructive paper on "Nonmalignant Intrathoracic Lesions Simulating Bronchiogenic Carcinoma" by Brewer, Jones, and Dolley,[1] there appears this statement about the treatment of hydatid cysts of the lung: "Since the best treatment for echinococcus disease is pulmonary resection, if the condition of the patient permits, pulmonary resection is the procedure of choice."

With this advice I heartily disagree, as do most Australian surgeons with experience in the treatment of hydatid cysts. Logan and Nicholson[2] have written in favor of lobectomy, and the indications for lobectomy have been discussed fully by Barrett.[3] In a recent paper[4] I gave my reasons for opposing many of Barrett's indications. Since then I have had twelve additional cases, and these have served to confirm my advocacy of conservative surgery for most hydatid cysts even when complications have already occurred before operation. Let me quote, from the summary of this paper,[4] the treatment that I recommended:

> When the lung is adherent to the chest wall:
>
> a. If the cyst is not infected, the contents are removed and the adventitia is closed.
>
> b. If the cyst is infected, the contents are removed, and the cyst space is drained. Should the pleural cavity be entered during the operation it must be securely closed before opening the adherent cyst.
>
> When the lung is not adherent:
>
> a. If the cyst is not infected, the pleural cavity is freely opened and protected, the contents of the cyst are removed, the adventitia is closed and sutured to the chest wall, and the pleural cavity is drained, with a water seal, through a stab wound.
>
> b. If the cyst is infected, the pleural cavity is opened and protected, the contents of the cyst are removed, the cyst space is drained and marsupialized, and the pleural cavity is drained through a stab wound.
>
> Lobectomy may be necessary for the following conditions:
>
> 1. Serious hemorrhage from the cyst space during a conservative operation.
> 2. Residual bronchiectasis, if symptoms warrant it.
> 3. Empty sac, if hemorrhage or infection warrant it.
> 4. When the diagnosis is uncertain.
> 5. Giant cyst.

I have now treated two infected cysts without drainage of the cyst space, just as if they had been uninfected, and in this I have differed from the advice just quoted. This is not to say that my earlier treatment was wrong, but only that it may be made still more simple. The records of these two cases follow.

From Sydney Hospital and the Royal North Shore Hospital.
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CASE 1.—L. T., a boy aged 13 years, complained of vague discomfort in the right side of the chest in September, 1947. Two months later he had several attacks of hemoptysis at short intervals, but in the following seven months he was apparently quite well. A radiogram taken during this period (April, 1948) showed an opacity in the apex of the right lower lobe (Fig. 1). In June, 1948, after falling from a horse, he had a large hemoptysis and became ill with a productive cough and fever. There were no abnormal signs to be found in the chest, but a radiogram now showed a crescent of air in the opacity (significant of rupture of the cyst into a bronchus) (Fig. 2). The Casoni test gave no reaction, and no hydatid elements were found in the sputum, but a white cell count showed 5 per cent eosinophils.

Under intratracheal anesthesia the thorax was opened through the posterior part of the eighth rib bed. The free pleural cavity was entered and a hard mass in the lung was found adherent to the chest wall just above the incision. The pleural cavity was shut off, a segment of the seventh rib was removed, and an incision through the adherent zone found pus and collapsed hydatid membrane. Most of the pus was aspirated and the remainder was removed along with the membrane. Examination with a spotlight showed several bronchial fistulas, and at the same time I noticed that the cyst space was becoming smaller as the surrounding lung expanded; at this stage the anesthetist aspirated some blood through the Magill tube. The cyst space was dried and powdered with penicillin, and then closed loosely with four interrupted linen thread sutures. The wound was swabbed with 5 per cent formalin and closed with interrupted sutures. Although the cyst was firmly adherent I put a tube through the ninth interspace into the pleural cavity to anticipate possible pleural soiling and to hasten re-expansion of the lung which was free everywhere except at the site of the cyst.

The patient made a quick recovery; for several days he was febrile and he coughed up some bloody sputum. The lung expanded promptly and the intercostal tube was removed on the seventh day. A radiogram taken on the eighteenth day showed an irregular opacity, and he was discharged free of symptoms (Fig. 3).

CASE 2.—J. M., a man aged 20 years, had a brisk hemoptysis after an injury to his chest. In the next seven months he had recurrent hemorrhages and gradually some purulent sputum was brought up between these attacks. His general condition remained good throughout, a point of diagnostic significance. Physical signs were indefinite and bronchoscopy showed nothing abnormal. There was a radiologic opacity in the apex of the left lower lobe (Figs. 4 and 5). At operation I resected a rib too low and the free pleural cavity was opened; there was a firm mass in the lower lobe adherent to the chest wall just above the incision. The pleural cavity was shut off and pus with collapsed membrane were removed from the cyst. The following steps of the operation were as in Case 1. The intercostal tube was removed on the fifth day and a radiogram on the fifteenth day was clear except for lack of translucency at the base (Fig. 6).

These two cases show clearly that after the cyst membrane and its purulent contents have been removed, the residual space and the surrounding lung may be so clean that they can be treated as being uninfected. I had felt that this was so earlier in my experience but had feared to dispense with drainage of the cyst space after pus had been found at operation.

I am now more than ever convinced that the barrier formed by the adventitia makes all the difference between an infected hydatid cyst and an ordinary lung abscess, and that, if there has been no soiling of the lung itself, closure of the cyst space is safe and proper. However, should there be the slightest doubt it is better to drain it.

In the following case I drained an infected adherent cyst without opening the free pleural cavity.

Fig. 1. Fig. 2. Fig. 3.

Fig. 1 (Case 1).—Cyst in apex of right lower lobe. (Confirmed by lateral view.)
Fig. 2 (Case 1).—Air space in cyst, due to rupture into bronchus.
Fig. 3 (Case 1).—Radiogram taken eighteen days after operation.

Fig. 4.

Fig. 5.

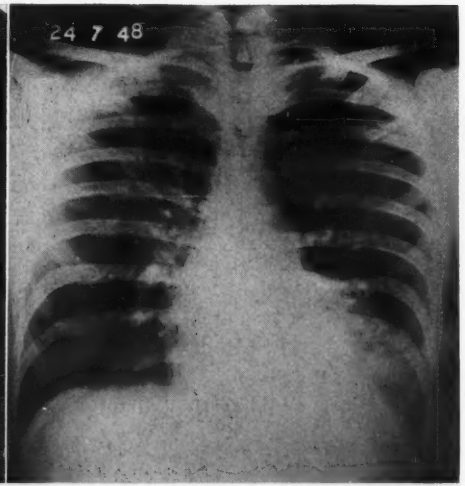

Fig. 6.

Fig. 4 (Case 2).—Cyst in apex of left lower lobe.
Fig. 5 (Case 2).—Lateral radiogram, showing cyst in apex of lower lobe.
Fig. 6 (Case 2).—Radiogram taken fifteen days after operation. There is incomplete aeration of the lower lobe.

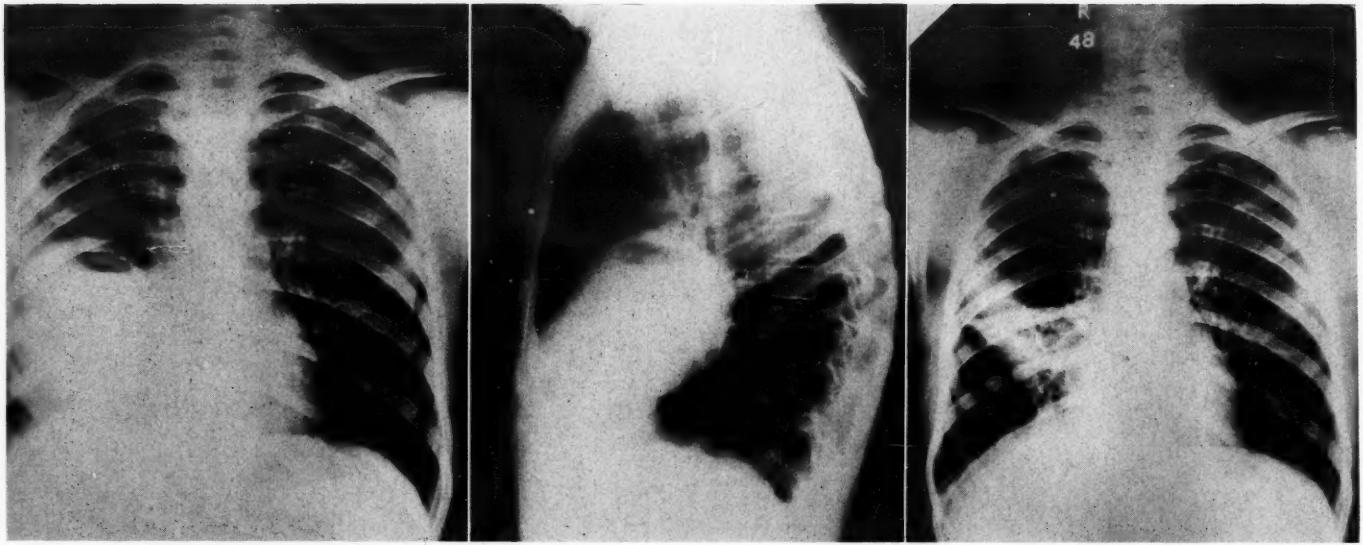

Fig. 7. Fig. 8. Fig. 9.

Fig. 7 (Case 3).—Cyst of middle lobe, or adjacent part of upper lobe; it has ruptured into the bronchus.
Fig. 8 (Case 3).—Lateral view corresponding to Fig. 7.
Fig. 9 (Case 3).—Radiogram taken two weeks after operation, showing irregular opacity and cavitation.

CASE 3.—J. C., a boy aged 15 years, gave a history of pleurisy on the right side in January, 1947. He soon recovered without any residual symptoms and remained well for the next eighteen months. Then he had an acute illness with fever and productive cough; some of the sputum was blood stained. Six weeks later he was admitted to the hospital, febrile and anemic with a productive cough and signs of consolidation of the right upper and middle lobes. He improved with chemotherapy and blood transfusion. No hydatid elements were found in the sputum, but the Casoni test was positive. A radiogram showed an opacity with a fluid level in the right middle lobe or adjacent part of the upper lobe (Figs. 7 and 8).

At operation I resected a segment of the fourth rib between the anterior axillary line and the costal cartilage. The cyst was found firmly adherent at this site and it was opened with a small scalpel. Offensive pus and collapsed membrane were removed. Some brisk hemorrhage followed this, but it soon stopped. (Hemorrhage after the opening of a hydatid cyst is sometimes so severe that lobectomy must be performed at once.)

The cavity was several inches in diameter and did not become appreciably smaller under observation; because of this, and because there was a larger bronchial fistula than usual, and because the pus was offensive, I deemed it safer to put in a tube, closing the rest of the wound around it. I passed an bronchoscope at the end of the operation and aspirated some bloody pus from the right bronchus. The patient made a good recovery; the tube was removed on the tenth day and two weeks later a radiogram showed an irregular residual space (Fig. 9). The patient was well and symptomless three months later.

It may be advisable to drain both the cyst space and the pleural cavity, as in the two following cases.

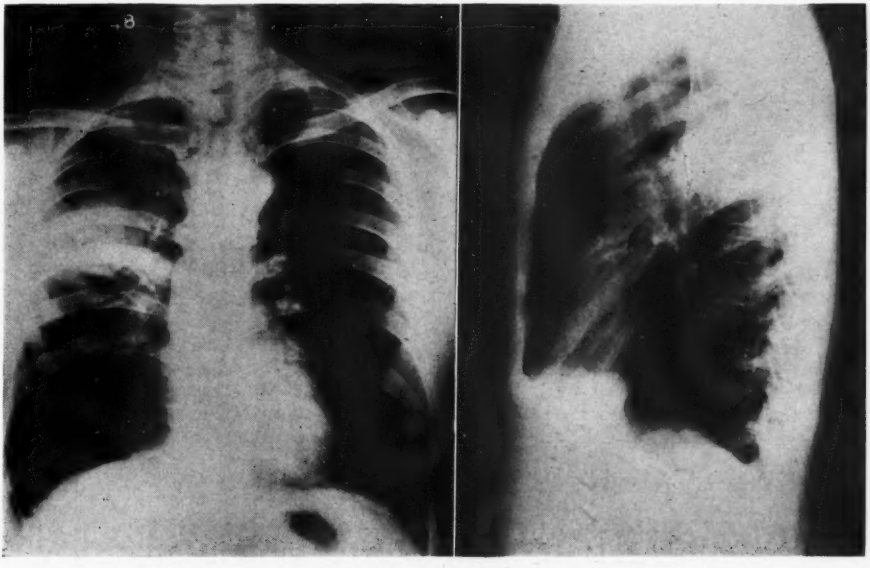

Fig. 10. Fig. 11.

Fig. 10 (Case 4).—Cyst in right upper lobe. Although it has ruptured into a bronchus there is no air space or fluid level.

Fig. 11 (Case 4).—Lateral view corresponding to Fig. 10.

CASE 4.—T. F., a man aged 53 years, had a "pneumonia" of the left side eighteen months before his present illness. He was now admitted to the hospital with a suppurating hydatid cyst. There were signs of hyporesonance and diminished air entry over a large part of the right upper lobe, and corresponding to these signs there was a rounded opacity in the radiogram, with surrounding infiltation (Figs. 10 and 11).

In spite of the suppuration his general condition was good. He had about one-half ounce of mucopurulent sputum daily in which no hydatid material could be found. He did not react to the Casoni test but the complement fixation test was positive. Clubbing of the fingers was noted.

At operation I removed a segment of the sixth rib from near the angle to just beyond the scapular line. The lung was seen to be moving under the rib bed, and I deliberately opened the pleura for exploration. I felt a firm mass in the upper lobe adherent farther forward than I had expected, under the fifth rib, near the posterior axillary line. I closed the incision in the sixth rib bed and then opened the adherent cyst from which the crumpled membranes and some debris were removed. Although the cyst space became smaller under observation, I decided to drain it because of some bleeding and because of a fairly large bronchial fistula. In retrospect, I think that it would have been quite safe to have closed the cyst space as in the first three cases. I also put a tube into the pleural cavity for sealed drainage. Although his general state remained good throughout, complete recovery was delayed because of slow re-expansion of the lung. However, within five weeks it had completely re-expanded and he was dismissed well.

CASE 5.—J. R., a girl aged 19 years, had coughed up some "frothy material" three weeks before admission to the hospital. Her condition was poor and she had severe dyspnea; the blood picture showed an eosinophilia of 25 per cent and the hydatid tests were positive. A radiogram showed a large air space in the left upper lobe with an opacity at the bottom (Figs. 12 and 13). During my absence a surgical colleague operated under unsatisfactory conditions, as the anesthetist refused to give the patient a general anesthetic. (From my experience I find that an intratracheal anesthetic is certainly preferable if not absolutely necessary unless one can be certain of finding the lung and cyst adherent.) Under local anesthesia he removed a segment of the fourth rib in the axilla and the free pleural cavity was opened: the cyst was found to be adherent posteriorly. A two-stage operation was decided upon and stitches were passed between the visceral and parietal pleuras to promote further adhesion, and gauze soaked in iodine was left in the wound. The next day the patient was ill and febrile. A radiogram showed that there was a hydropneumothorax below the cyst (Fig. 14). Air was aspirated from the pneumothorax and nine days after the operation a tube was inserted (through the fourth interspace in the axilla) into the pleural cavity. I first saw the patient thirty days after the operation, when she had general constitutional upset, with remittent fever and purulent sputum. At this stage I opened the thorax through the eighth rib bed. The lower lobe was found collapsed against the mediastinum and there was a little fluid in the pleural cavity. The cyst in the upper lobe was adherent, and the membranes and much offensive pus were removed. A tube was put into the cyst space and into the pleural cavity and both were connected to a water-seal drainage system. The wound was closed loosely about the tubes. Her condition was precarious throughout this operation, but she soon showed signs of improvement. The lung gradually became fully aerated and expanded, and a radiogram taken nine months after the last operation showed a well-expanded lung (Fig. 15).

The next two cases show what conservative surgery can do even in the face of the serious complication of rupture of the cyst into the pleural cavity, causing pneumothorax and infection. "Hydatid pneumothorax," though a well-known possibility, is not common if one may judge from the small number of reported cases. It may occur apparently spontaneously or it may fol-

low some trauma such as direct injury, a straining effort, or coughing. Occasionally it is a quiet event with few symptoms at first, but generally, as with other forms of spontaneous pneumothorax, there may be sudden and dramatic

Fig. 12. Fig. 13.

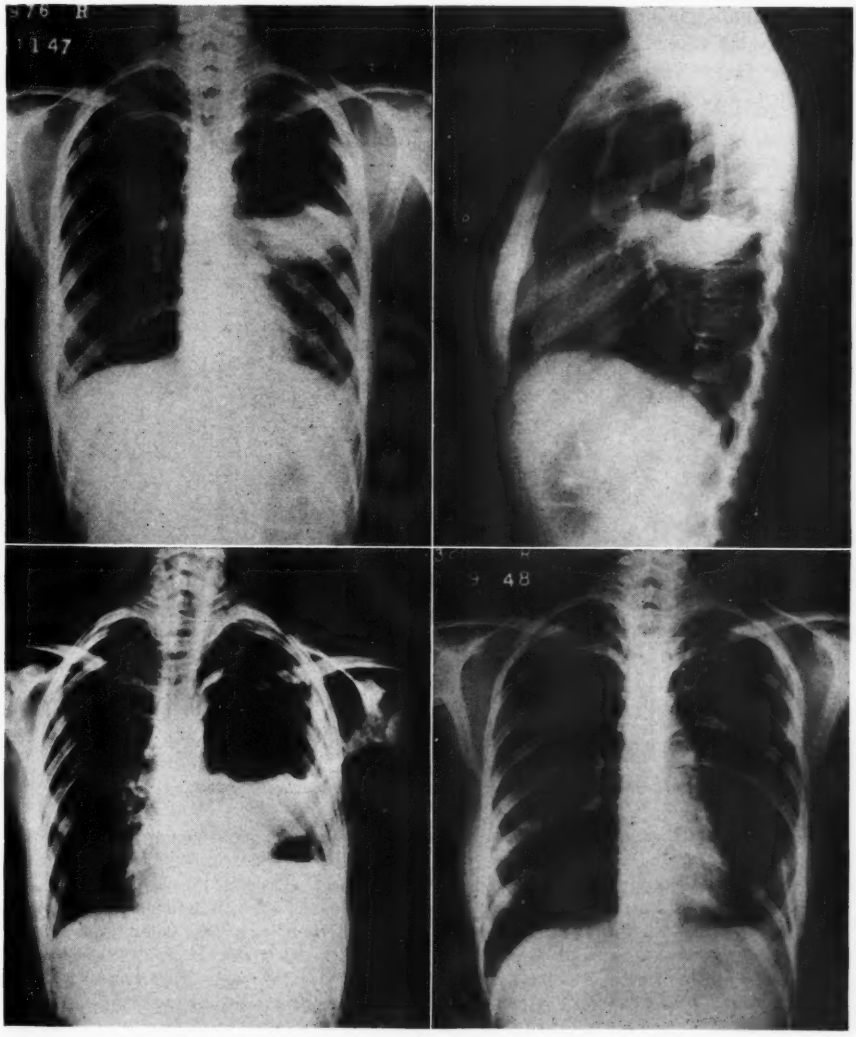

Fig. 14. Fig. 15.

Fig. 12 (Case 5).—Hydatid cyst of left upper lobe. The cyst has ruptured into the bronchus and most of the fluid has been coughed up. The collapsed hydatid membranes lie at the bottom of the air-filled cyst.

Fig. 13 (Case 5).—Lateral view corresponding to Fig. 12.

Fig. 14 (Case 5).—There is now a hydropneumothorax below the cyst.

Fig. 15 (Case 5).—Radiogram taken nine months after last operation. The lung has completely re-expanded.

symptoms which may be more overwhelming than usual because of the added symptoms of anaphylaxis. If operation is delayed, pleural infection is almost inevitable, and still later there may be secondary hydatid cysts in the pleural cavity. It is clear that prompt operation is essential as soon as the pneumothorax occurs, both to save life and to avoid further complications; the ruptured membranes and all fluid and debris must be removed, any obvious bronchial fistulas should be closed, and the pleural cavity should be drained by a water seal. In the two cases here reported pyopneumothorax was already present before the patients came into my ken.

CASE 6.—A man, aged 56 years, coughed up some fluid and soon afterward had a spontaneous pneumothorax followed by fever and dyspnea and failure of the lung to re-expand. A radiogram showed a collapsed and airless lung with a complete pneumothorax. Thoracoscopy three months later showed that the collapsed lung was covered with a layer of white fibrin. At this stage he had one-half cup of yellow sputum daily; no hydatid elements were

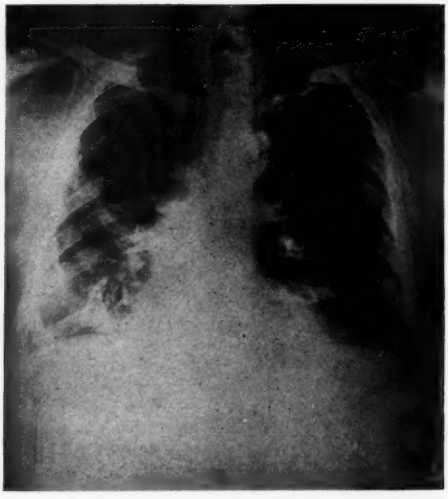

Fig. 16 (Case 6).—Radiogram taken one and one-half years after operation.

found in it. The Casoni test was negative, but the complement fixation was positive. Bronchoscopy and bronchography gave no positive information. It was decided that exploratory thoracotomy was necessary both for diagnosis and treatment. I opened the chest wall through the fifth rib bed and noted thickening of the parietal pleura. The whole lung was collapsed and covered with thick fibrin. I set out to decorticate the lung and a good "peel" was found in the costovertebral groove, but further forward the fibrin layer was closely attached to the lung and the decortication was incomplete. Just above the diaphragm I came upon a deep excavation in the lung with several bronchial fistulas, and after a little more manipulation I found collapsed hydatid membrane lying between the diaphragm and the base of the lung. The excavation in the lung was evidently the original site of the cyst before its rupture into the pleural cavity.

I decorticated the lung as thoroughly as possible and, when the bronchial fistulas had been closed, it aerated slightly when the anesthetist raised the pressure. Two drainage tubes were left in the pleural cavity, one in front and one behind; they were both connected to a

water seal, and the main wound was closed. Later, suction was applied to the drainage tubes. The lung re-expanded slowly after prolonged drainage and Fig. 16 is the radiogram taken one and one-half years later; although the general condition of the patient improved he had repeated small hemoptyses in the following year. Bronchoscopy and bronchography showed no abnormality. It is now over two years since operation, and he is well except for moderate dyspnea on exertion and occasional staining of the sputum.

CASE 7.—H. L., a woman aged 45 years, had a "pneumonia" in December, 1947. After recovery from this, a radiogram showed a hydatid cyst of the left lung. Just before admission to the hospital the cyst ruptured into the pleural cavity and bronchus. Signs of pneumothorax were found, and soon there was evidence of sepsis in the lung and the pleural cavity. A radiogram showed the pneumothorax with a fluid level, and just above and medial to it part of the lung was visible in which there was a small cavity with a fluid level; the mediastinum was displaced to the right (Fig. 17). She reacted to the Casoni and complement

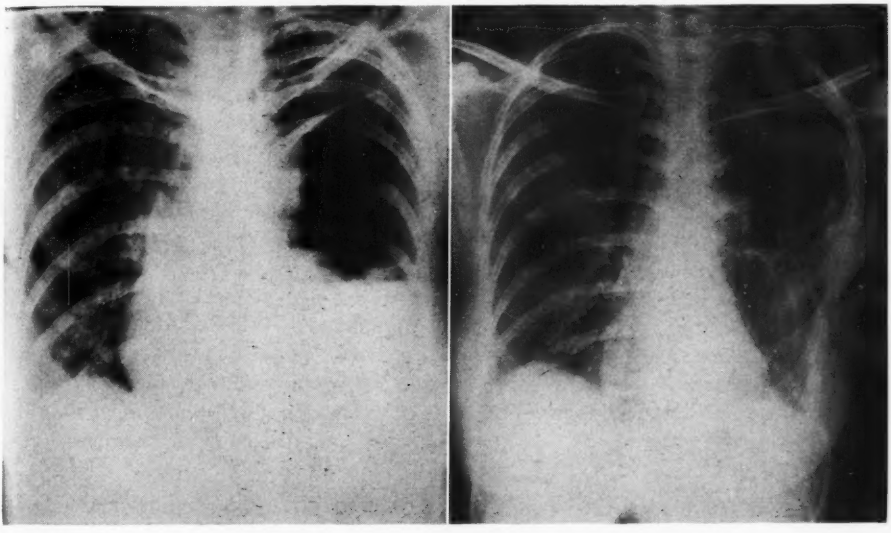

Fig. 17. Fig. 18.

Fig. 17 (Case 7).—Large pyopneumothorax, with displacement of heart to the right. Just above and medial to the fluid level in the pleural space there can be seen, in the collapsed lung, the ruptured hydatid cyst with a fluid level.

Fig. 18 (Case 7).—Radiogram taken seven months after the final operation (for plastic closure of the residual empyema cavity).

fixation tests, but no hydatid material was found in the sputum. The first operation was simple closed drainage of the empyema through a small rib resection (sixth) in the midaxillary line. This gave prompt relief and within two days the upper lobe had become fully expanded; the lower half of the lung field remained opaque and the mediastinum became central. She still had some purulent sputum, but it was much less than on admission. One month after this operation thoracotomy was done; six inches of the eighth rib were removed and an empyema cavity was opened, containing a little pus and crumpled hydatid membrane. The upper lobe was firmly adherent to the chest wall, the lower lobe moved on respiration, and two bronchial fistulas were seen; these were closed with several silk sutures. The main wound was closed except posteriorly where a tube was put in for drainage. Two months later her general condition was good, but there was still a chronic empyema space and one or both of the bronchopleural fistulas had reopened. Another operation was performed for un-

roofing of the empyema cavity and the filling of it with a large flap from the latissimus dorsi muscle and two intercostal muscles. This operation served to close the defect and she was soon well except for pain in the course of the lower intercostal nerves. Fig. 18 is the radiogram taken seven months later.

CONCLUSIONS

Most hydatid cysts of the lung can be adequately treated by conservative surgery, consisting of removal of the cyst membranes and fluid and drainage of the pleural cavity or of the cyst space or of both, according to the conditions found. Even when complications have already occurred, conservative surgery usually suffices. Lobectomy is needed for a small number of hydatid cysts.

Editor's Note: The illustrations present no postoperative bronchograms. When the author was questioned about his failure to include such bronchograms, he stated that he had not made them routinely because all but two of the patients recovered quickly and apparently completely. In the two cases in which the recovery was not so rapid, bronchograms were made and were found normal.

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BRONCHIECTASIS ASSOCIATED WITH ANOMALY OF THE RIGHT PULMONARY VEIN AND RIGHT DIAPHRAGM

Report of a Case

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THE increased frequency with which resection of lobes of the lung is performed at present must mean that congenital anomalies within the thorax are being more frequently encountered. However, there are few reports of such anomalies in the medical literature. This is unfortunate, since a clear knowledge of the possible anomalies and their significance is important to the surgeon, both in understanding the pathogenesis of disease and, technically, for the safety of the patient. The case presented in this report illustrates a combination of anomalies, one of which was probably responsible for the patient's disease process. The other made surgery technically more difficult.

It is of interest to note that there is an increased incidence of pathologic processes in lungs containing congenital anomalies of one type or another. Gruenfield and Gray[1] have discussed the common congenital malformations of the lungs, pointing out the increased incidence of pulmonary tuberculosis and bronchiectasis in the azygos lobe. Womack and Graham[2] have analyzed the incidence of bronchiogenic carcinoma in lungs with congenital abnormalities. Adams and Churchill[3] have likewise considered the more frequent occurrence of bronchiectasis in patients with situs inversus. The present case does not fit into the classification of the usual abnormalities, and since it offered a puzzling picture preoperatively as well as at the time of operation, it seems worthy of presentation.

CASE REPORT

J. L. (R-13879), a 24-year-old man, an accountant, was admitted to the Veterans Administration Hospital, West Roxbury, Mass., on July 28, 1947, complaining of a cough of five years' duration. The cough was most marked in the morning and was productive each day of one-half cupful of yellowish, nonfoul but occasionally blood-flecked sputum. Two years prior to admission, the patient had had a hemoptysis of one cupful of bright red blood. There was also a history of exertional dyspnea after climbing one flight of stairs.

Physical examination was noncontributory except for the presence of diminished breath sounds and fine crepitant râles at the right base anteriorly and posteriorly. The left lung was normal.

X-ray examination of the chest (Fig. 1) showed that the heart was displaced to the right but was normal in rotation, and with the aortic arch in its normal position. In the lateral view, a linear shadow was seen extending posteriorly from the apex of the diaphragm to the posterior part of the chest wall. These roentgenograms were interpreted as demon-

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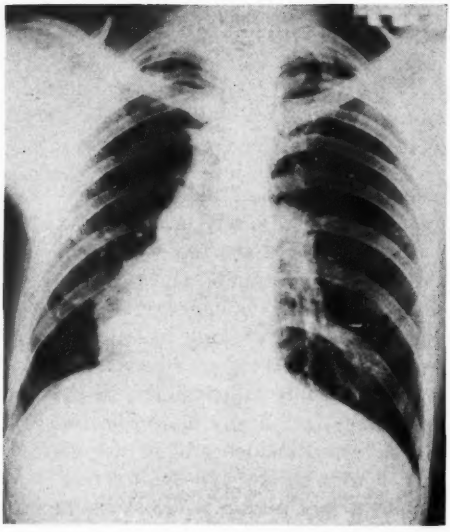

A.

B.

Fig. 1.—*A*, Posteroanterior roentgenogram of chest. *B*, Right lateral roentgenogram of chest on admission.

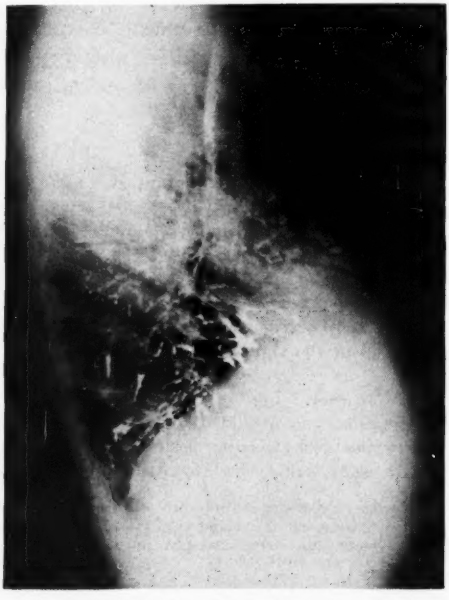

Fig. 2.—Right lateral bronchogram of chest.

strating a partially atelectatic lower lobe with increased aeration of the middle lobe. The previously mentioned linear shadow was interpreted as the fissure betwen the middle and lower lobe.

At bronchoscopy, the mucous membrane of the right lower lobe was slightly redder than normal but otherwise examination was not remarkable. A bronchogram demonstrated a normal left lung. On the right side there was cylindrical bronchiectasis of the anterior basic division of the right lower lobe. The other basal segments appeared decreased in size with crowding of the bronchi as seen in the lateral view (Fig. 2). It was felt that the symptoms could be accounted for by pathology in the right lower lobe, and operation was advised.

At the time of thoracotomy, the right lower lobe was found to be divided into two portions by an extra leaf of the diaphragm which arose from the dome of the normal right diaphragm and extended laterally and posteriorly to join the chest wall two inches above the normal diaphragmatic insertion. The two portions of the lower lobe were connected posteromedially where this accessory leaf did not reach the chest wall. Through this connecting isthmus of the lung, the vascular and bronchial supply of the trapped segment passed. Beneath this "backpocket" segment was a normally attached, complete diaphragm (Fig. 3).

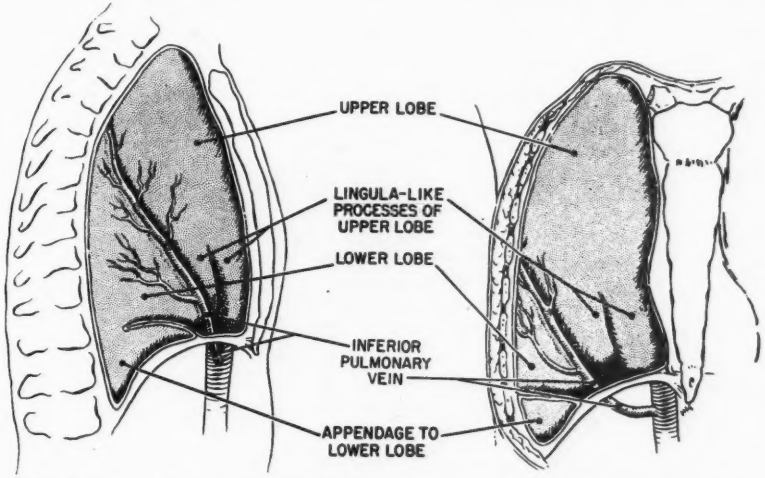

Fig. 3.—Lateral and anterior illustration of venous and diaphragmatic anomaly.

There was no true fissure between the middle and upper lobes. What should have been the middle lobe consisted of two lingular processes extending over the right border of the heart, which lay to the right of its usual position.

In the fissure between this upper and the anomalous lower lobe was discovered a large vein which proved to be the inferior pulmonary vein (Fig. 3). It extended downward and anteriorly through the fissure, receiving branches from the lower and upper lobes, to penetrate the diaphragm at its mid-point just anterior to the junction of the normal and anomalous leaves. On opening the diaphragm, this vein was found to run medially along its inferior surface and to empty into the inferior vena cava. A normal superior pulmonary vein was found emptying into the left auricle in the usual position.

The right lower lobe was removed by individual ligation technique, the tributaries of the inferior vein being divided without sacrificing the vein. The accessory superior segment of the diaphragm was excised, eliminating the inferior pocket. The chest wall was then closed with interrupted silk sutures, and temporary intercostal catheter drainage was established.

Postoperatively the patient's course was uneventful, and he left the hospital without symptoms on the eighteenth postoperative day.

At follow-up four months later, there was a slight cough. The patient had gained fifteen pounds and there was good expansion of the right upper lobe as shown by roentgenogram. Mild dyspnea persisted. Further follow-up, one year after operation, showed that the patient's course had been uneventful. There was still some cough, especially after respiratory infection, but generally the patient had gained weight and felt well. Mild dyspnea still persisted. There had been no further hemoptysis.

DISCUSSION

Anomalies of the pulmonary vein are probably very common, even though the number of reports of such are few. Reports by Evans,[4] Harris and Lewis,[5] and Brown[6] indicated the embryologic background of such anomalies. As described by Harris and Lewis,[5] the pulmonary veins are the eventual persisting forms of an original vast indifferent plexus lying in relation to the primitive foregut of its lung bud. This gut plexus drains by two veins into the primitive sinus venosus, which later becomes the left auricle. The plexus has numerous anastomoses with both the anterior and posterior cardinal vein. Of the two main veins entering the primitive left auricle, the superior eventually becomes the main pulmonary veins. The inferior of the two original veins becomes the hepatic portion of the future inferior vena cava. Persistence of this latter trunk into adult life explains the anomaly encountered in our patient, namely, communication of the pulmonary venous system with the hepatic

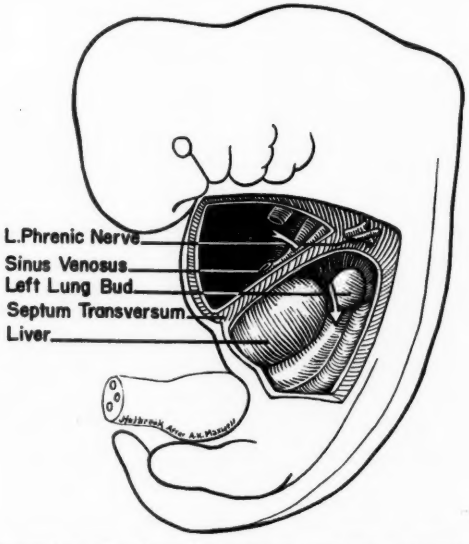

Fig. 4.—Schematic illustration of embryo showing the lung bud developing in the defect posterior to the edge of the septum transversum.

portion of the vena cava. Brown[6] in his report described the finding, during anatomic study, of an anomaly of the inferior pulmonary vein similar to the one described in this report.

Descriptions of anomalies of the diaphragm have been largely confined to a discussion of deficiencies with eventual herniation. No report was found describing an anomaly such as seen in this patient. However, a possible explanation suggests itself from the embryology of the diaphragm. The diaphragm is formed largely from the septum transversum and the pleuroperitoneal membrane. In early embryonic life, the septum transversum is in a high position and during development it continues to recede caudally until it reaches the position of the adult diaphragm. In the early stages, the lung buds growing out from the foregut project into the forepart of the pleuroperitoneal cavity. Owing to descent of the dorsal end the septum transversum, the lung buds come to lie above the septum. It would appear possible that at an early stage in development, less rapid progress of the septum transversum caudally might trap a portion of the lung below it. Then the developing pleuroperitoneal membrane, which should meet the advancing edge of the septum transversum would, of necessity, develop beneath the trapped lung, reaching the septum transversum ventral to its advancing edge. The result would be a "backpocket" defect such as we found in this patient with the trapped lung. The possibility that such might occur is illustrated in the accompanying diagram (Fig. 4).

SUMMARY

1. A case is presented demonstrating numerous congenital abnormalities within the thorax. These were dextroposition of the heart, absence of a true middle lobe, and a peculiar anomaly of the right diaphragm consisting of a reduplication of its posterior portion. There was also an anomalous inferior pulmonary vein which penetrated the diaphragm to empty into the inferior vena cava.

2. We believe that greater attention should be given to congenital anomalies within the thorax since they may influence the presence of disease in the patient and may complicate surgical therapy.

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GIANT-CELL TUMOR OF RIBS

A Case Report and Survey of Literature

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THE exact number of cases of primary giant-cell tumors of the ribs is difficult to determine due to the lack of adequate pathologic descriptions. In previous surveys by Hedblom[1] in 1933, Samson and Haight[2] in 1935, Sommer and Major[3] in 1942, Dorner and Marcy[4] in 1948, there have been nine cases of giant-cell tumors of the thoracic cage reported dating from Fort's[5] case in 1914 to Janes'[6] case in 1939. Of these only eight have been reported in literature. Neil's, as described by Samson and Haight,[2] was presented by personal communication. Codman described a specimen of giant-cell tumor of costal origin at the Warren Museum, Harvard Medical School. Christensen likewise described a case of costal giant-cell tumor, but all records have been lost.

Giant-cell tumors of the thoracic cage have no great significance, per se; however, due to their infrequency as compared to the incidence of giant-cell tumors of other parts of the bony framework, the subject becomes of medical interest.

As well as can be determined from literature, this case to be presented is the largest giant-cell tumor of the thoracic cage, which may well be due to its duration of approximately eight years. A biopsy made six years previously revealed it to be a giant-cell tumor.

CASE REPORT

The patient, a 49-year-old, white man, a confectioner, was first seen on Aug. 28, 1948, with the complaints of (1) a "large tumor" on the left side of the chest, (2) pain over the region of the tumor which was not relieved by the usual analgesics, (3) weakness, (4) slight fever, and (5) general lack of well-being.

Present Illness.—The patient stated that in December, 1940, he noticed the appearance of a tumor on the left lower part of the chest. The tumor made its appearance about two months after an injury due to a fall down a flight of stairs. The tumor gradually enlarged and caused considerable pain and discomfort. After the tumor had been present for eighteen months the patient was admitted to a hospital where removal was attempted in June, 1942. Removal at that time was abandoned due to excessive hemorrhage and only a biopsy was obtained. The diagnosis by the pathologist at that time was benign giant-cell tumor of bone.

The mass continued to grow slowly and became more painful. In 1943, the patient received deep x-ray therapy with a total of 2,400 roentgen units in nine treatments given. Again in 1948 he received 3,060 roentgen units in seventeen treatments given over a period of six months. There was no regression in size of the tumor and no relief of pain. The severity of the pain had gradually increased.

Physical examination revealed a well-developed, well-nourished, white man, 49 years of age. The temperature was 101° F., pulse 104, respiration 24, the blood pressure was 180 systolic and 110 diastolic. The only finding of note was the tumor, which was a large, hard, protruding mass, located on the left lower part of the chest and extending from the

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fifth rib in the midaxillary line to the iliac crest and from two inches lateral to the vertebral column posteriorly to the unbilicus anteriorly. The skin over and immediately surrounding the tumor was of a dull, reddish-purple color and was shiny and indurated; several areas of ulceration were present. The surface of the tumor was very irregular and loculated due to the presence of multiple cystic areas of varying sizes. There was considerable tenderness to palpation and the tumor was fixed to the underlying structures. Several of the cystic areas were aspirated. Three hundred cubic centimeters of red-tinged, serous fluid were obtained, thus relieving the pain slightly.

X-ray and fluoroscopic examination revealed a large area of slightly increased density over the lower left part of the chest extending from the fifth rib anteriorly to the crest of the ilium, and to the mid-portion of the abdomen. The ninth, tenth, eleventh, and twelfth ribs were involved; no excursion of the diaphragm could be made out. An upper gastrointestinal x-ray examination revealed the stomach and small bowel to be displaced markedly to the right.

It was decided an attempt at removal should be made despite the patient's poor general condition. The approach decided upon was transthoracic, in an attempt to get behind the tumor and enucleate it in toto.

The patient was admitted to Mt. Carmel Hospital, Columbus, Ohio, on Aug. 29, 1948.

Laboratory Work.—Red blood count was 3,690,000 and white blood count, 6,000. Aug. 30, 1948, urinalysis showed 250 mg. per cent of albumin, plus fine granular and cylindrical casts; 2 plus bacteria, 2 plus mucus. Aug. 31, 1948, albumin was 400 mg. per cent, cylindrical casts, 3 plus. Total proteins on Aug. 30, 1948, were 5.8 mg. with 4.1 to 1.7 albumin-globulin ratio. Serology was negative.

Operation.—On Aug. 31, 1948, the patient was operated upon under endotracheal anesthesia and the tumor was removed.

A left posterolateral thoracotomy incision was made above the tumor, through the bed of the eighth rib which was resected subperiostally. The diaphragm was incised; the peritoneal cavity was entered and by manual palpation the tumor was found to extend beyond the umbilicus. A second incision was made from the anterior end of the transthoracic incision to the crest of the ilium and a skin flap definitely free of tumor tissue was mobilized by sharp dissection. The skin was ulcerated and markedly indurated due to the previous x-ray therapy and probable local pressure necrosis. The descending colon and stomach were separated from the tumor from below upward by sharp and blunt dissection. The tumor was rotated laterally from its medial surface and finally removed in that manner with blunt and sharp dissection. Due to the involvement of the chest wall, the anterior and lateral portions of ribs twelve, eleven, ten, and nine, together with their intercostal bundles, were removed.

After removal of the tumor mass, the peritoneal cavity was closed without difficulty. The diaphragm was then repaired and a new attachment was made posteriorly to the stumps of ribs nine and eight, and anteriorly to the costochondral junction at about the level of rib five, bringing about a complete closure of the diaphragm at a considerably higher level than normal, the pleural cavity having been drained with two No. 30 catheters attached to a water trap.

A cigarette drain was left in the angle near the crest of the ilium. Approximately 3,000 c.c. of citrated blood were given during the procedure per ankle cannula although very little bleeding occurred. A postoperative bronchoscopy was done. The patient was returned to his room in good condition.

Because of the extensive tissue changes in the skin, débridement had to be carried out at a later date and when an attempt at healing by granulation was unsuccessful, the area was covered with a split-thickness skin graft.[18] The general condition of the patient was good, the skin graft being completed.

Pathology.—The surgical specimen consisted of a massive tumor weighing 3¼ kg. (7½ pounds). It measured 20 by 15 by 15 cm., and was lobulated and very irregular in shape. Attached to the upper portion were four pieces of rib.

The tumor was firm in consistency in most places but one large egg-shaped mass on the outer surface measuring 15 cm. in diameter was cystic to palpation. The color of the tumor was mottled yellow, red, and light tan. In some areas there was blackish discoloration due to old hemorrhage. The solid portion on cutting was granular and the color of putty. On the inner surface numerous small cystic areas were palpated. On cutting, the cystic areas were filled with thin, watery, reddish discolored fluid and the walls were covered with multiple loculated areas, some of which were filled with fibrin and blood pigment.

Microscopic examination of sections taken from the tumor revealed a pleomorphic picture. The cells were spindle shaped and fusiform and there were numerous multinucleated giant cells. Diffusely scattered throughout were numerous fibroblasts and large areas of hyalinized fibrous connective tissue. There was a thin hyalinized fibrous connective tissue capsule which was well delineated from the tumor. In the more active areas there were numerous small blood vessels and nodules of siderofibrotic character. There was no evidence of malignancy. The final diagnosis was giant-cell tumor with degeneration and fibrosis.

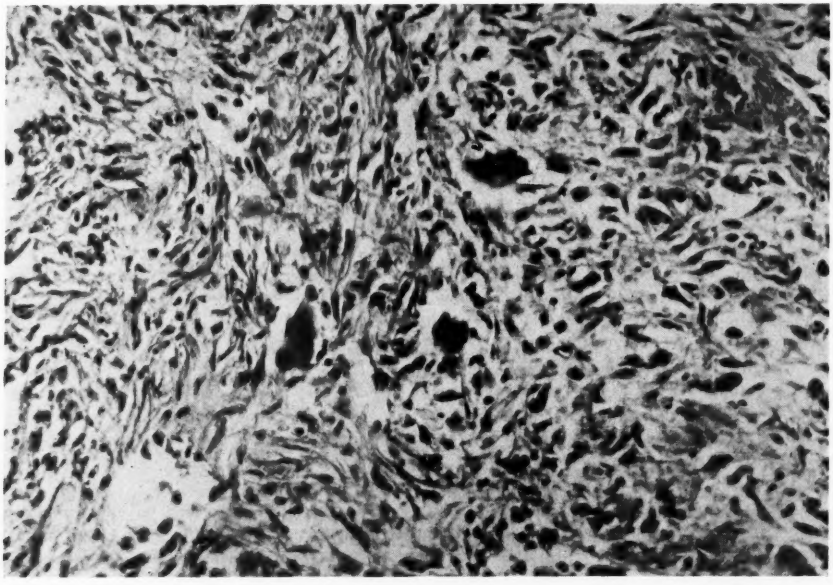

Fig. 1.—Photomicrograph of removed tumor.

DISCUSSION

A summary of the collected cases of giant-cell tumors of ribs is presented in Table I.

The usual clinical history given by patients with giant-cell tumors of costal origin is similar to that obtained in giant-cell tumors in other locations; namely, the sequence of trauma, pain, and tumor extending over a period of from two to sixteen months. Trauma has been fairly well established as a definite etiological factor in the relation of onset of the disease, although in this series of ten cases, trauma was denied in four instances.

TABLE I

TABLE I

AUTHOR	AGE (YR.)	SEX AND RACE	SITE	SIZE	SYMPTOMS	DURATION	TREATMENT	RESULTS	HISTOLOGIC DIAGNOSIS	TRAUMA	REMARKS
Fort[5]	11	M, W	1st rib, clavicle ant.	5 × 3 × 3 cm.	Tumor	8 mo.	Excision post. of 1st rib, clavicle sternum, 1914	No deformity, well 17 yr. later	Benign giant-cell tumor	No	
Gatewood[7]	14	F, W	Left 12th rib, post.	5 × 3 × 3 cm.	Pain, tumor, scoliosis	1 yr.	Excised followed by radium therapy, 1921	Well 13 yr. later	Benign giant-cell tumor	Yes	
Peirce[8]	17	M, W	Left 10th rib, post.	6 inch	Pain, tumor, loss of weight (20 lbs.), dry cough	9 mo.	X-ray with excision of ribs 8 to 12, left, 1930	Died 40 hr. postoperative	Chrondro-osteogenic (benign) sarcoma in giant-cell tumor of rib infiltrating	Yes	Only reported case with malignant changes in giant-cell tumor of rib
Hedblom[1]	38	F, W	Right 8th rib, post.	7 × 2½ cm.	Pain, tumor	3 yr.	Excision, 1922	Well 11 yr. later	Benign giant-cell tumor	Yes	Injury 12 yr. before tumor evident; intercostal ne tomy (8th) 1919 gave relief
Holland[9]	48	M, W	5th rib, costochondral junction	4 × 3 × 3 cm.	Tumor	6 mo.	Excision, 1923	Well 11 yr. later	Benign giant-cell tumor	No	
Neil[2]	9	M, W	Left 4th at angle	Small	Pain	2 mo.	Biopsy, radium, 1923	Well 11 yr. later	Benign giant-cell tumor	Yes	
Samson and Haight[2]	31	F, W	Rt. 2nd rib, costochondral junction	3.5 × 2 × 2 cm.	Tumor	2 yr.	Excision, 1935	Well 5 yr. later	Benign giant-cell tumor	No	
Hilt[10]	15	F, W	Left 5th rib at angle	3.5 × 3 × 3 cm.	Pain, tumor	6 mo.	Biopsy, 1935; x-ray, 1935	1 yr. and 10 mo. later tumor smaller	Benign giant-cell tumor	Yes	
Janes[6]	49	M, W	Rt. 7th rib, post.	8 × 5 cm.	Pain	10 mo.	Excision, 1936	Died 2½ yr. later of bronchial ca.	Benign giant-cell tumor	No	
Authors,[1] case	55	M, W	Left 11th rib with thoracic wall invasion and abdominal extension	20 × 15 × 15 cm.	Tumor, pain, ulceration	8 yr.	Biopsy, 1942; x-ray, 1943, 1948; excision, 1948	Well 5 mo. later	Benign giant-cell tumor	Yes	Nine x-ray treatments, 1943; 17 x-ray treatments, 1948; no apparent regression

The rarity of giant-cell tumors of costal origin, as suggested by Geschickter and Copeland,[12] is perhaps due to the extremely small and relatively inactive epiphyses of the ribs. In this series, four of the tumors were posterior, two were at the angle, two were at the costochondral junction, and two involved an entire rib.

The size of the tumor does not seem to be in proportion to the duration of symptoms, although in the authors' case definitive treatment would have been considerably easier had surgery been carried out earlier.

Due to the inadequate series, it is extremely difficult to draw any definite conclusions as to the proper treatment. The duration between onset and treatment of the tumor does not appear to influence the success of the definitive treatment unless, as in Peirce's[8] case, malignancy is involved. Direct excision was used in five cases, irradiation only in two cases, excision followed by irradiation in one case, and irradiation followed by excision in one case.

According to Meyerding,[13] radiologists have discovered that benign and low-grade malignant giant-cell tumors are radiosensitive. While x-ray therapy may be effective in some cases it apparently had no effect on the authors' case, either in controlling pain or diminishing the size of the tumor. Although giant-cell tumors are typically benign, recurrence after surgical excision is not rare and scattered reports in the literature are continually suggesting the possibility of metastases.[13] This fact has not been borne out in giant-cell tumors of costal origin but as the series is small, no definite conclusions can be made. Invasion of soft parts is not uncommon and was demonstrated in four of the cases: Fort,[5] Peirce,[8] Hedblom,[1] and the authors' case. The invasive power of giant-cell tumors may be an explanation of the occasional recurrence. However, in spite of possible malignant changes that may occur or the possibility of recurrence, the prognosis following adequate treatment has been excellent.

SUMMARY

1. A case of giant-cell tumor of costal origin is presented. Its size and duration significantly demonstrate the benignity of these tumors. That these tumors are uncommon is evidenced by only nine other authentic cases being found in the literature.

2. Treatment is variable; excision, irradiation, or combination of these methods give apparently satisfactory end results.

3. It is the belief of the authors that the treatment of choice is early surgical excision, while the tumors are still small, and before they invade the soft parts. In so doing, recurrence and malignant changes will be largely avoided.

4. Providing the therapy has been adequate and malignant changes have not yet taken place, the prognosis is excellent.

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THE USE OF TANTALUM PLATE WHEN RESECTING LARGE AREAS OF THE CHEST WALL

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THE resection of large areas of the chest wall which are the seat of malignant disease has a much wider application at the present time than was heretofore possible. This type of operation, which is more frequently associated with pulmonary neoplasms and which may also include the removal of one lobe or the entire lung, exemplifies the present status of excision surgery in this region.

Cancer of the lung with extension to the pleura and ribs has in the past been one of the criteria of inoperability. This concept is being revised in the light of our present knowledge that peripheral lesions have a tendency to local invasion rather than to early spread to the mediastinum as is the case with those more centrally located.

When the disease is so situated that the adjacent chest wall may be removed with the tumor, hope is offered to a relatively large group of patients whose plight was formerly considered hopeless. An additional favorable factor lies in the fact that one of the major lobes can more frequently be preserved than is the case with lesions of the main-stem bronchi.

The excision of a large segment of chest wall gives rise to certain problems that must be considered and dealt with. Paradoxical respiration must be guarded against when there is a mobile mediastinum, and when dealing with large defects this cannot always be satisfactorily controlled by external appliances or pressure dressings. If the hazards of the postoperative period can be surmounted, there nevertheless remains a flabby chest wall which is subject to motion with each respiratory effort.

When, in addition to the bony support, all of the overlying soft tissues must be removed, as was true in Case 1, satisfactory coverage must also be provided.

The following cases illustrate methods of overcoming these problems. In addition to eliminating the immediate postoperative dangers of a weakened chest wall, permanency is created in the defective area.

CASE REPORTS

Case 1.—C. T., a 63-year-old white woman, was admitted to Rhode Island Hospital on Feb. 5, 1947, complaining of a gradually enlarging mass in the upper part of the sternum. She first noticed discomfort and a small nodule in this region in June, 1946. She consulted her physician who advised observation and that she return if there was any enlargement of the nodule. The mass remained reasonably static until early in December, when it appeared to increase in size rather rapidly, but she failed to consult her physician again until two weeks prior to admission to the hospital.

From the surgical service of the Rhode Island Hospital.
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The patient had always been well with the exception of "arthritis" in several joints which had been troublesome for about eleven years. Recently she had noticed a slight increase of dyspnea on exertion.

Examination revealed a well-developed and well-nourished white woman who appeared to be about the stated age. There was a mass about 7 cm. in diameter which was firmly fixed to the anterosuperior portion of the sternum and extended down to the third interspace. It was fixed to the bone beneath but not attached to the underlying skin. It was smooth, hard, and somewhat tender. The heart and lungs were essentially normal. The blood pressure was 190/90.

Urine examination revealed a specific gravity of 1,220, and a trace of protein. The hemoglobin was 14.0 per cent, and the white blood count 8,250 with 69 per cent polymorphonuclears. The blood glucose was 102, total protein 7.1, blood calcium 9.8, and blood urea nitrogen 9. The globulin was not increased. Inorganic phosphorus was 3.2; alkaline phosphatase was 27.1. The Hinton test was negative.

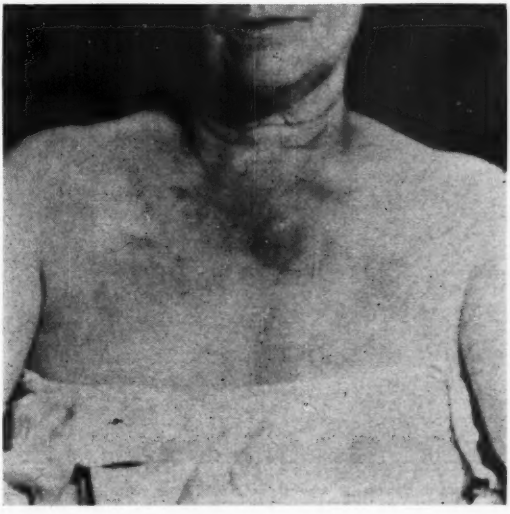

Fig. 1 (Case 1).—The tumor is seen bulging into the soft tissues over the manubrium.

X-ray examination of the skull was negative. The entire spine and pelvis showed marked changes in several of the dorsal and lumbar vertebrae and all the pelvic bones, characteristic of advanced Paget's disease. There was a collapse of the body of the fourth lumbar vertebrae with slight expansion in both the anteroposterior and lateral directions. The cervical spine showed moderate hypertrophic changes but no other bony pathology. Both lower extremities also disclosed marked involvement of the femurs and upper articular ends of the tibiae due to Paget's disease. The right tibia also showed a slight thickening of the cortex on the lateral aspect of the mid-shaft. Films of the sternoclavicular region revealed a marked rarefaction and expansion of the manubrium of the sternum. The anterior portion was irregular and showed destruction with invasion or infiltration of the underlying soft tissues. The impression was that of advanced Paget's disease, involving several bones. There was a destructive malignant bone lesion of the manubrium and sternum, probably sarcoma on the basis of associated Paget's disease.

On Feb. 7, 1947, a biopsy of the tumor was taken, and the pathologic examination revealed osteogenic sarcoma of the sternum.

Operation was performed on March 11, 1947. Endotracheal cyclopropane anesthesia was used.

The operation consisted of wide block excision of the tumor. In order to accomplish this it was necessary to resect portions of the third, second, and first ribs on both sides, as well as the inner aspect of both clavicles, and to transect the sternum at the level of the third interspace. In the neck, the muscle attachments to the clavicle were divided through a transverse incision across the suprasternal area. The tumor was found to bulge into the anterior mediastinum, where it was adherent to the innominate veins, from which it was separated by sharp and blunt dissection. It was then possible to remove this entire segment of the anterior chest wall with the tumor. During the dissection, the pleura on either side was reflected so that neither pleural cavity was entered. A tantalum plate, which had previously been perforated to allow for the escape of serum, was cut to fit the major portion of the defect and sutured with No. 38 wire to the cut ends of the second and third ribs on either side and to the sternum.

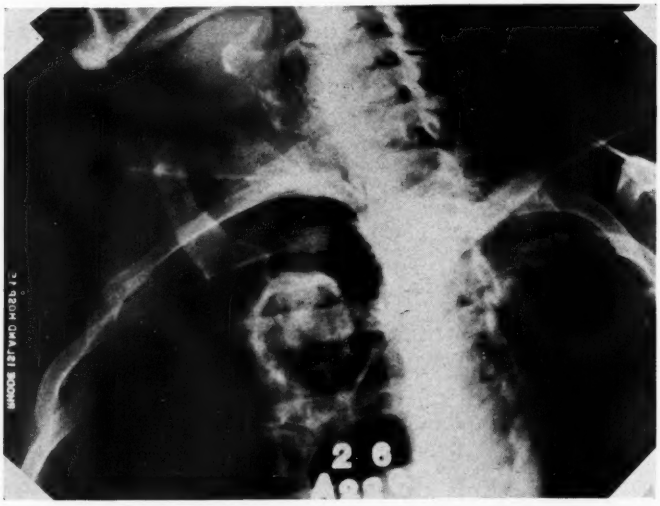

Fig. 2 (Case 1).—Roentgenogram of tumor. An oblique view showing destruction of manubrium and invasion of the sternoclavicular joints.

A large pedicle flap was then raised which included the entire right breast with the underlying pectoralis major and its fascia. This was rotated upward and sutured into the defect. One small rubber tissue drain was inserted beneath the pedicle in the lower portion of the wound. By undermining, it was possible to close the major portion of the area from which the pedicle had been raised, leaving only a small area to be skin grafted. During the course of the operation 1,000 c.c. of blood was given. The patient's condition was excellent at the completion of the procedure.

The pathologic report was as follows: The specimen consisted of an elliptical piece of skin measuring 14.5 by 7.5 cm., in the center of which was a scar 2 cm. in length. Attached to the skin was a section of sternum 10 cm. in length, right and left clavicles measuring 4.5 and 7 cm., respectively, and both right and left, first, second, and third ribs were attached to the sternum. Skin and subcutaneous tissue were removed in toto. There was a swelling 5 by 4.5 cm. which included the left clavicle and a small portion of the right sternoclavicular junction. It was gray white, firm, and covered by a thin, fibrous capsule. On the undersurface of the sternum there was an additional swelling 4.5 by 2 cm. It was brownish red, soft, and rubbery and covered by a thick, fibrous capsule. Cut sections revealed a spherical mass 4.5 by 4 cm. The surface showed pearly, white, soft, gelatinous areas with streaks of gray-white to white-yellow calcified material. There were

scattered hemorrhagic areas in some of the gelatinous material. This tumor extended into the sternoclavicular joints, but did not involve the clavicles proper. It occupied two-thirds of the manubrium. The gross diagnosis was osteogenic sarcoma.

The microscopic description showed that of the six sections, four were composed of hyperplastic cartilage, fibrocartilage, and scattered newly formed bone trabeculae and periosteal tissue. There was a marked overproduction of immature cartilage and precartilagenous and perosseous tissue. All layers except the bony trabeculae showed more than normal activity. One section was made up of striated muscle and surrounding fibroadipose tissue. It showed edema and scattered leucocytic infiltration with a few polymorphonuclears in some areas. A section of skin and subcutaneous tissue showed a marked fibroblastic activity beneath a thin squamous epithelial layer. The layer was infiltrated with leucocytes and endothelial leucocytes with a very dark-staining cytoplasm. Often these cells contained multiple nuclei up to large foreign body giant cells. The deep edge

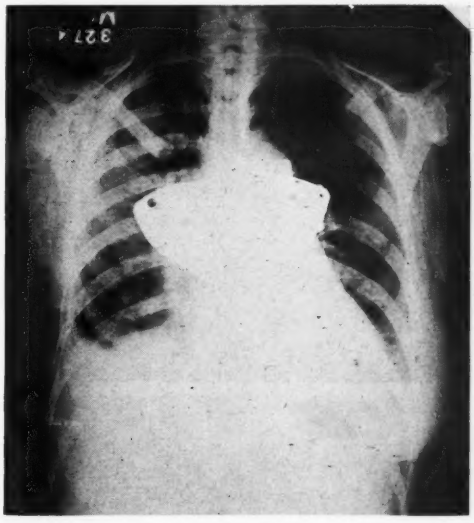

Fig. 3 (Case 1).—A postoperative roentgenogram showing the tantalum plate in position.

of the section showed the wall of an abscess cavity containing numerous macrophages and foreign body giant cells. The diagnosis was osteogenic sarcoma of sternum (osteochondrosarcoma).

The patient's postoperative course was uneventful. There were no pulmonary complications, and she was out of bed on the third day.

Oxygen was given for the first twenty-four hours as a routine measure. There was considerable serous drainage during the first week which subsequently became much less but persisted in small amounts. The patient was kept in the hospital until April 14, 1947, so that she could be carefully observed, after which she was followed in the Tumor Clinic. Her general condition showed very definite and progressive improvement, but as had been anticipated, a moderate amount of serous drainage persisted.

She was readmitted to the hospital on June 24, 1947, for the purpose of removing the tantalum plate. It was decided that enough time had elapsed for a satisfactory inflammatory membrane to have developed. The operation merely consisted of elevating one side of the flap, cutting the wire sutures, and sliding out the plate. With the flap lifted up, it was interesting to note that the foreign body reaction had created a firm, unyielding inflammatory membrane which allowed no motion with respiration. The mem-

Fig. 4 (Case 1).—Section through tumor and sternum. Note destruction of manubrium.

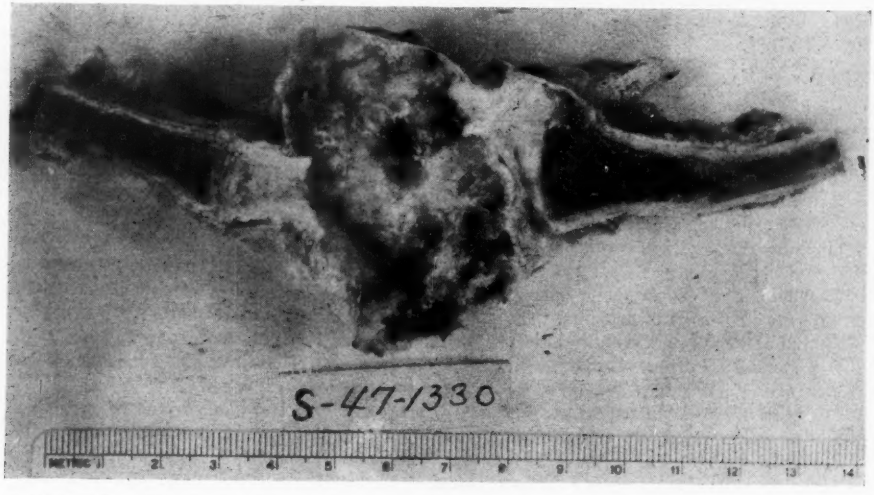

Fig. 5 (Case 1).—Section through tumor and clavicles showing invasion of sternoclavicular joints.

brane had formed on the anterior and posterior surfaces of the plate, but of the two, the mediastinal surface appeared to be the more rigid. The incision was closed with interrupted silk.

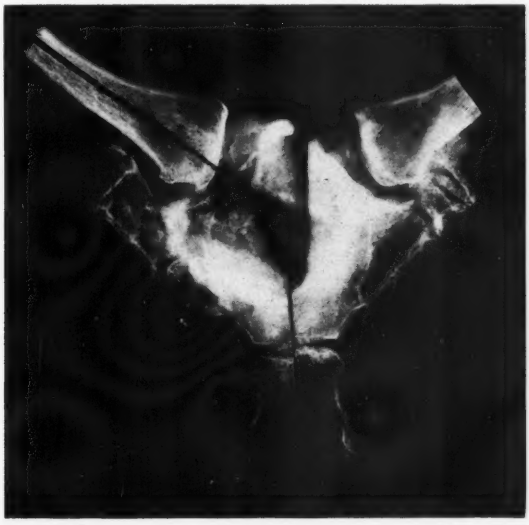

Fig. 6 (Case 1).—Roentgenogram of specimen indicating the extent of the resection.

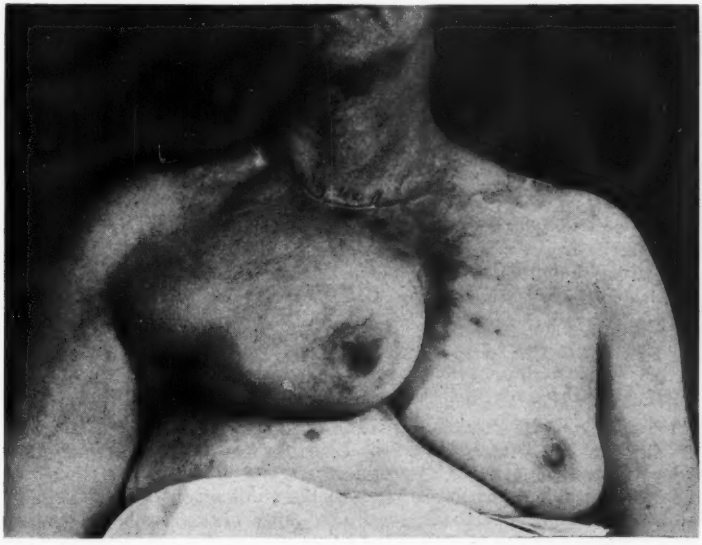

Fig. 7 (Case 1).—Final result showing defect closed with a pedicle flap which includes the right breast and the underlying pectoralis major muscle with its fascia.

After removal of the plate there was prompt healing of the wound. The patient has been followed at regular intervals at the Tumor Clinic, the last visit being on Jan. 12, 1949, at which time she was free of symptoms and carrying on her usual activities.

CASE 2.—W. M., a 72-year-old white man, was admitted to the Rhode Island Hospital on April 29, 1948, with a complaint of cough and pain in the left anterior part of the chest of about one month's duration which had been growing steadily worse. He had had no expectoration or hemoptysis, and although he complained of fatigue, he had noticed no loss in weight.

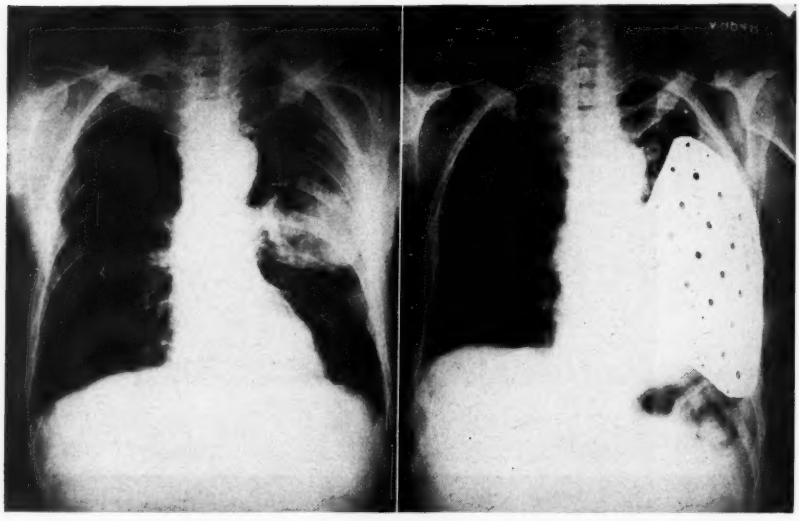

Fig. 8. Fig. 9.

Fig. 8 (Case 2).—Preoperative roentgenogram of chest.
Fig. 9 (Case 2).—Postoperative roentgenogram with tantalum plate replacing the bony defect.

Examination revealed an elderly man who appeared chronically ill. There was moderate sclerosis of the peripheral arteries, but his heart was essentially normal to physical and electrocardiographic examination. Blood pressure was 156/84. The breath sounds and tactile fremitus were slightly decreased throughout the left side of the chest. There was moderate dullness noted in the mid-chest region anteriorly and posteriorly, and fist percussion on the left caused slight pain. No râles were present. General examination was otherwise not remarkable.

Red blood count was 5.01, hemoglobin 15, and white blood count was 15,150 with 65 per cent polymorphonuclears. Blood urea nitrogen was 17, glucose 81, and protein 6.6. Hinton test was negative. Bronchoscopy on April 20, 1948, was entirely negative. No tumor was seen, and no abnormal secretions were noted.

The report of the x-ray examination of the chest on April 19, 1948, was as follows: the roentgenogram showed a roughly rounded, irregular density in the mid-portion of the left lung field, extending from the seventh to the ninth ribs posteriorly. On the lateral view the mass appeared to be situated well posteriorly, apparently in the apex of the left lower lobe. There was definite erosion and beginning destruction of the inferior border of the posterior axillary portion of the seventh rib. Other portions of the lung were clear. The cardiovascular outlines were normal. There was no shifting of the trachea, heart, or

mediastinum. The diaphragms were normal. There was no effusion. The conclusions were that the findings were characteristic of a malignant neoplasm of the left lung with extension to an involvement of the adjacent seventh rib and pleura.

The patient was placed on aerosol penicillin four times a day, and the operation was performed on May 6, 1948.

A right-angled incision was made from the fourth to the eighth ribs paravertebrally and along the eighth interspace to the posterior axillary line. The soft tissue flap was elevated and the ribs exposed at their spinal junctions, after which the fifth, sixth, seventh, and eighth ribs were divided with the intervening, intercostal bundles. A long incision was then made through the fourth interspace, and the pleural cavity opened. It was now possible to make a careful exploration of the lung and the tumor. The latter was found to be adherent to the sixth, seventh, and eighth ribs but had no pleural attachments beyond this area. The incision through the fourth interspace as well beyond the limits of the tumor from above. It appeared to arise from the apex of the lower lobe and was adherent to the lower surface of the upper lobe posteriorly. There was no obvious invasion of glands around the lung root. The mediastinal pleura was incised, and the artery and vein to the lower lobe were dissected out, doubly ligated, and divided, after which the bronchus was isolated and cut across and closed with four interrupted sutures of No. 30 cotton. The mediastinal pleura was then closed with interrupted No. 30 cotton sutures. With the lower lobe divided, it was then possible to rotate it laterally, and a clear exposure of the attachments of the tumor to the chest wall could be obtained. The excision was completed by incising through the eighth interspace and dividing the fifth, sixth, seventh, and eighth ribs in the posterior axillary line and including the lower portion of the upper lobe in the excision. The entire specimen was then removed en bloc. The large defect in the chest wall was closed with a tantalum plate, which was fashioned to fit the defect and was fixed in place with wire sutures after drilling the fourth and ninth ribs at intervals of 2 cm. and the stumps of the cut ribs proximally and distally. One cigarette drain was placed on the surface of the plate and brought out at the lower portion of the wound which was closed in layers. The upper lobe was re-expanded as the chest wall was closed. One catheter was inserted in the dependent part of the thorax.

The pathologic report was as follows: the specimen consisted of a lower lobe with attached portions of four ribs. The diaphragmatic surface was not unusual. The bronchial tree in the lower portion was dilated with thickened mucosa and transverse markings. Binding the four ribs firmly to the surface of the lung was a tumor mass 7 by 4.5 by 5 cm. which had been incised. The surface peripherally was gray, hard, and granular, while centrally the tissue was soft and necrotic. The bones were thinned and apparently involved by tumor. Leading from the amputated margin into the central cut surface of the tumor was a thin-walled small bronchus. The bronchus had lost its continuity and disappeared within this tumor mass. No other bronchi were seen to traverse the tumor tissue. On section the tumor had a gray-tan, granular surface. The pulmonary arteries were not involved in the tumor mass. The portion of tumor invading the bone was firm, rubbery, and elastic. Areas containing greenish-yellow puriform material measuring up to 4 mm. in greatest diameter were seen scattered throughout the tumor mass. The remaining lung tissue had a yellow-tan surface covered by a wrinkled pleura, which showed numerous red, fibrous tags. It appeared collapsed and on section presented a smooth, nonporous, nonrepitant surface. A portion of another lobe which measured 4 by 2 by 1.5 cm. was adherent to the overlying rib and adjacent lobe.

Section of lung showed replacement of lung parenchyma by cords of anaplastic epithelial cells which had squamous characteristics. There was occasional pearl formation. At the periphery of the tumor areas the alveoli contained clumps of similar appearing cells. Tumor was also seen within a periarterial lymphatic. Section of rib showed invasion by and replacement by infiltrating cords of similar appearing cells. The diagnosis was epidermoid carcinoma of the lung (bronchiogenic) with invasion of pleura and ribs.

The patient's course during the operation was excellent. There was no abnormal blood pressure fall and no suggestion of shock; 1,500 c.c. of blood were given. He was allowed up on the second postoperative day.

His course was complicated by atelectasis of the left upper lobe with associated cough and temperature elevation. Bronchoscopy was carried out, and a considerable amount of thick mucopurulent secretion was aspirated from the trachea and the left upper lobe bronchus, after which the lobe became well aerated and the symptoms relieved.

There was considerable serous drainage from about the plate which later became seropurulent. This was associated with temperature rise, and it became evident that drainage was inadequate. He was taken to the operating room on June 15, 1948; one side of the flap was elevated and the plate removed. Examination revealed a moderate amount of organized exudate within the space and a firm inflammatory wall on both its surfaces. The wound was closed with one cigarette drain to its dependent portion.

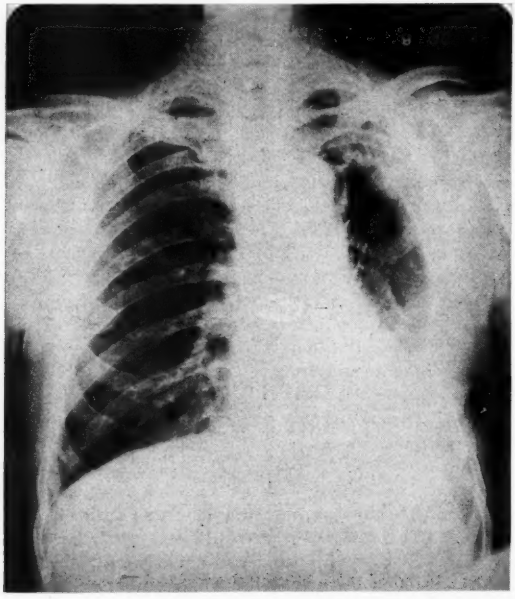

Fig. 10 (Case 2).—Roentgenogram taken Sept. 30, 1948. Note the marked thickening in the area previously occupied by the plate.

Definite improvement continued from this point on. This was rather slow at first, but the temperature subsided, and the drainage at the time of discharge on July 17, 1948, was only slight and was nonpurulent. There has been complete absence of chest wall pain, appetite is good, and he has continued to gain weight. Careful follow-up study was made, and he was last seen on Jan. 3, 1949. He had gained twenty-eight pounds since the hospital discharge and was entirely symptom free. The chest wall was firm and entirely without motion on respiration.

CASE 3.—J. H., a 58-year-old white man, was admitted to the Rhode Island Hospital on May 13, 1948, with a chief complaint of severe pain in the right side of the chest. He began to have pain in the right anterior part of the chest and right axilla six months prior to admission. It began rather suddenly in November, 1947, and grew gradually worse, and became constant. He was admitted to another hospital for observation in April, 1948. He had lost considerable weight and the pain had become so severe that large amounts of sedation

were required each day. There was no complaint of cough and no history of expectoration or hemoptysis. Roentgenogram of the lung fields was negative, and there was no evidence of destruction of the ribs.

He had had a mild cerebral accident about seven months previously. At that time some weakness of the left part of the face, left arm and leg were noted, but there had been no progression of these signs.

Examination revealed a white man who showed evidence of marked weight loss. There was a tumor palpable between the spine and the medial border of the right scapula about 6 by 5 cm. It was firm and irregular in outline and appeared to be firmly attached to the chest wall at about the level of the fifth and sixth ribs. It was slightly tender to palpation. Examination of the lungs revealed normal breath sounds and no dullness, and only slight limitation of expansion on the right. The heart sounds were normal, and the blood pressure was 110/60. There was slight facial asymmetry due to a left facial weakness. Knee jerks on the left were diminished. Babinski sign was negative, and no sensory changes were present. A slight weakness of the left leg was noted but no paralysis. Apart from general evidence of weight loss, physical examination was otherwise normal.

Laboratory work on entry showed a red blood count of 3.76, a normal urine, and a hemoglobin of 12.1 Gm. The blood urea nitrogen was 24, glucose 77, and the blood protein 4.7 mg. per 100 c.c.

X-ray examination of the chest revealed no infiltration of the lung fields and no destruction of ribs.

Because of the great severity of the patient's pain it was decided to resect the tumor which appeared to arise from the chest wall, although a primary source in the lung with extension through the intercostal spaces was considered.

Operation was carried out on May 19, 1948. A vertical paravertebral incision was made from the third to the eighth rib, well beyond the periphery of the palpable tumor, and deepened to expose the transverse processes, the third, fourth, fifth, and sixth of which were divided, as were their corresponding ribs and intercostal bundles. The incision was then extended laterally over the seventh rib, which was subperiosteally resected, and the pleural cavity entered. The lung was adherent to the parietal pleura but easily freed up to the region of the tumor where it was firmly adherent to the chest wall in an area about 5 cm. in diameter. The tumor and adjacent chest wall were then free at the posterior and inferior portions of the dissection. The outlined triangular area of skin and subcutaneous tissues was then elevated along with the scapula to expose the ribs in the upper part of the hemithorax.

The pleura was then opened along the line of the second interspace after which the third, fourth, fifth, and sixth ribs were divided in the posterior axillary line with the intervening intercostal bundles and pleura. The severed area of chest wall and the tumor were then completely free with the exception of their attachment to the lung. This appeared to be very superficial, and it could not be definitely demonstrated that there was invasion of lung tissue. However, it seemed wisest to excise a liberal amount of lung tissue rather than to run the risk of entering the tumor. A wedge of lung tissue was therefore included with the specimen which was then removed, and the defect closed with interrupted silk sutures. There was no suggestion of lung invasion at any other point. A palpable gland in the mediastinum was removed and found to be benign.

A tantalum plate which had previously been perforated at several points was cut to fit the bony defect and sutured into place with No. 28 wire. This was accomplished by drilling holes in the periphery of the plate and in the adjacent ribs. The plate was bent to conform to the natural convexity of the thoracic cage in this region. The soft tissues of the chest wall were then closed over the plate with one cigarette drain to the dependent portion of the wound.

The pathologic report was as follows: The specimen consisted of portions of four ribs and apparently included the transverse processes of four vertebrae with attached fibroadipose and muscle tissue. Involving three of the ribs near the vertebral end, there was a firm,

pinkish-tan, seemingly localized tumor measuring 1.5 cm. in greatest diameter which blended at its margins with the surrounding tissue. On section the tumor retained a sharp cut edge. It had a homogeneous mucosa-appearing surface.

Attached to the inner rib aspects there was a greenish-gray portion of lung measuring 5 cm. in its greatest diameter. The sections contained carcinoma that apparently originated in the lung and extended to involve the pleura and chest wall. The histology varied. In one section of lung tissue a small bronchus was involved. In some sections the cells lined alveolar spaces, giving an adenoid appearance. These cells were largely squamous in type, but in a few places became tall and columnar, and secreted mucus. There were also numerous small solid groups of squamous cells. Because of the predominance of the squamous histology it was decided to place the neoplasm in that category. The diagnosis was epidermoid carcinoma (squamous cell) of the lung, with extension to pleura and chest wall.

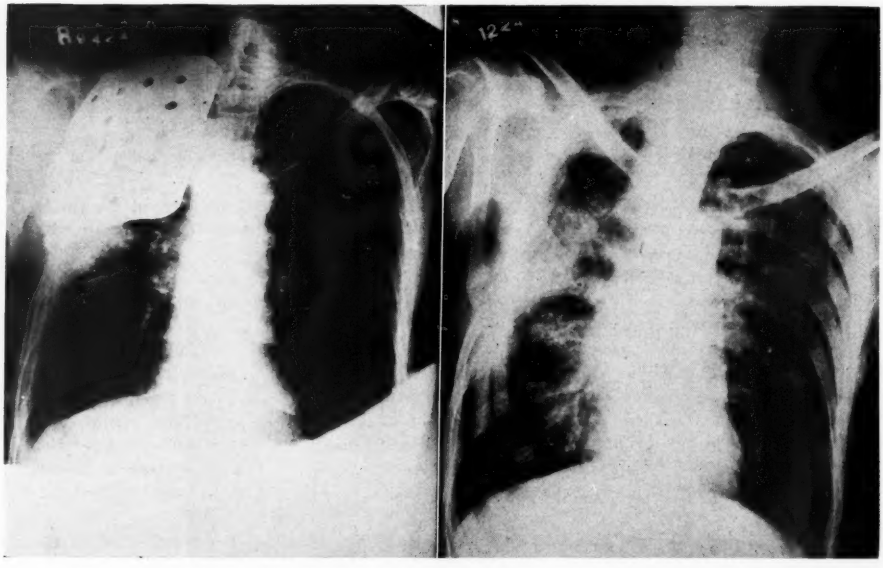

Fig. 11. Fig. 12.

Fig. 11 (Case 3).—Showing tantalum plate in position. Its size indicates exactly the extent of the resection.

Fig. 12 (Case 3).—Postoperative roentgenogram taken July 9, 1948.

The patient made an uneventful recovery from the operation. There was never any evidence of shock, and in spite of the wide resection of the chest wall there was complete stabilization of the right hemithorax. The patient was allowed out of bed on the third postoperative day. The most striking feature of the postoperative phase was the complete absence of pain. In spite of the fact that practically all sedation was discontinued, the patient was completely comfortable. There was a moderate amount of serous drainage from the wound which later became seropurulent. This was associated with some elevation of temperature, and on June 17, 1948, he was taken to the operating room, the skin flap elevated, and the tantalum plate removed. There was a moderate amount of localized infection on both sides of the plate which appeared to be due to superficial erosion from the lung surface. There was marked rigidity of the tissues which had been adjacent to the plate. After removal of the plate, one cigarette drain was inserted at the dependent portion of the wound. The fever subsided, and during the remainder of his hospital stay there was only slight serous drainage. In spite of the complete absence of pain and the

satisfactory appearance of the chest wall wound, the patient showed no evidence of weight gain. There appeared to be a gradual decline in his general condition in spite of a fair appetite, absence of pulmonary symptoms, and ability to be out of bed daily. He was discharged from the hospital on July 16, 1948, and follow-up study was made at home by his family physician. He died on Sept. 12, 1948.

COMMENT

The preceding cases, although presenting individual problems, all required extensive removal of the supporting framework of the thoracic cage. Unless favored with mediastinal fixation this may result in difficulties which can be serious in the immediate postoperative period. Attempts to neutralize the adverse effects of a swinging mediastinum, such as may be observed following extrapleural thoracoplasty, by means of external appliances or by packing and strapping of the wound have not always been successful. In cases where it is necessary to remove the periosteum with the ribs no stiffening of the chest wall can be hoped for, and although recovery from operation may occur, a flabby and mobile chest wall invariably follows. The use of tantalum plate in the manner described has obviated these difficulties.

Multiple perforations were made in the plate in one patient (Case 1), who was the first to be operated upon. This was so successful that the same type of plate was used in Cases 2 and 3, where a somewhat different problem was presented. In the latter instances the lung expanded against the plate, and in both cases an abnormal amount of reaction occurred. The reason for perforating the plate in Case 1 was to allow for drainage of serum from the anterior mediastinum. Adequate drainage from the pleural cavity can, of course, be effected by means of a catheter in a dependent position.

In the future a smooth plate will be used when dealing with lesions involving the lung. This is particularly applicable when only a lobectomy has been done and the remaining lobe must expand against the plate.

When in addition to removal of the bony structures a wide resection of the skin and other soft tissues must be carried out, large lateral pedicle flaps are most useful. On the anterior surface in the female, the breast is ideal for this purpose, but regardless of the location of the donor site the pedicle should include muscle and fascia in order to give proper stability. By undermining, the major part of this defect can be closed and the remainder covered with a split-skin graft. The inflammatory wall created by the foreign body reaction completes the formation of a rigid chest wall. The final result bears a close resemblance to that obtained following a Schede type of thoracoplasty for chronic empyema.

It is believed that the dramatic relief of pain in Case 3 which allowed this patient to live out his remaining days in comparative comfort, justified the operation.

SUMMARY

Three cases are presented in which tantalum plate has been used to avoid complications which occur when a large section of the chest wall has been removed. This has had the effect not only of overcoming postoperative difficulties but, in addition, has given stability to the area of excision.

RECONSTRUCTION OF THE ANTERIOR THORACIC WALL

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THE sacrifice of large portions of the anterior thoracic wall incident to the removal of malignant neoplasms is required in selected cases. These cases include (a) primary neoplasms arising in the bony thoracic wall, (b) recurrent mammary cancer invading the bony thorax, and (c) primary carcinoma of the breast infiltrating the thoracic wall at or near the costal cartilages or lateral aspect of the sternum, where protection from the thoracic wall is not afforded by the interposition of the pectoral muscles.

Although the past decade of progress in surgical technique, physiology, and anesthesia has permitted radical resections of the thoracic wall as well as viscera, few reports have dealt with methods by which these extensive wounds may be repaired. It is the purpose of this report to describe a method which has been satisfactorily utilized in the reconstruction of the anterior thoracic wall.

Relatively small defects in the anterior chest wall can be closed in a number of ways, and among the most satisfactory is the method described by Maurer and Blades[1] which furnishes bony stabilization to the defect. Watson and James[2] have described the use of fascia lata grafts in the repair of large defects, but these must be covered by large pedicled flaps of skin, for split-thickness grafts can scarcely be expected to survive on fascial patches which are, themselves, free grafts without blood supply. Maier[3] has discussed the use of the opposite breast as an aid in the closure of large defects. Pickrell, Kelley, and Marzoni,[4] in an excellent review of this problem, used pedicled flaps of various design, and in one case in which such a flap was not feasible they applied a split graft directly upon the pericardium. The following procedure which utilizes a fascia lata graft, a completely mobilized latissimus dorsi muscle, and split-thickness skin grafts appears to have advantages not possessed by the foregoing methods.

Excision of the Lesion.—Standards governing the removal of any neoplastic process apply to the excision of these lesions. They include removal of all the neoplasm en bloc, together with an adequate margin of uninvolved tissues. The lymph nodes draining the area occupied by the neoplasm must be removed, if possible. If the operation is being done for recurrent carcinoma of the breast, all the axillary lymph structures will have previously been removed, and this area need not be invaded the second time. Recurrence in the axilla or supraclavicular area generally constitutes inoperability, and palliation by roentgen therapy is preferable. If the lesion is located over the lateral margin of the sternum, the internal mammary vessels with accompanying lymph structures should be removed.

Resection of lesions infiltrating the bony thorax requires removal of the full thickness of the thoracic wall and rarely will a portion of less than two ribs suffice. In general, a block of chest wall requiring the segments of three, four,

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or five ribs is required, and frequently substantial portions of the sternum must be sacrificed as well. Such a defect frequently extends from the sternum to a point near the anterior axillary line (Fig. 2).

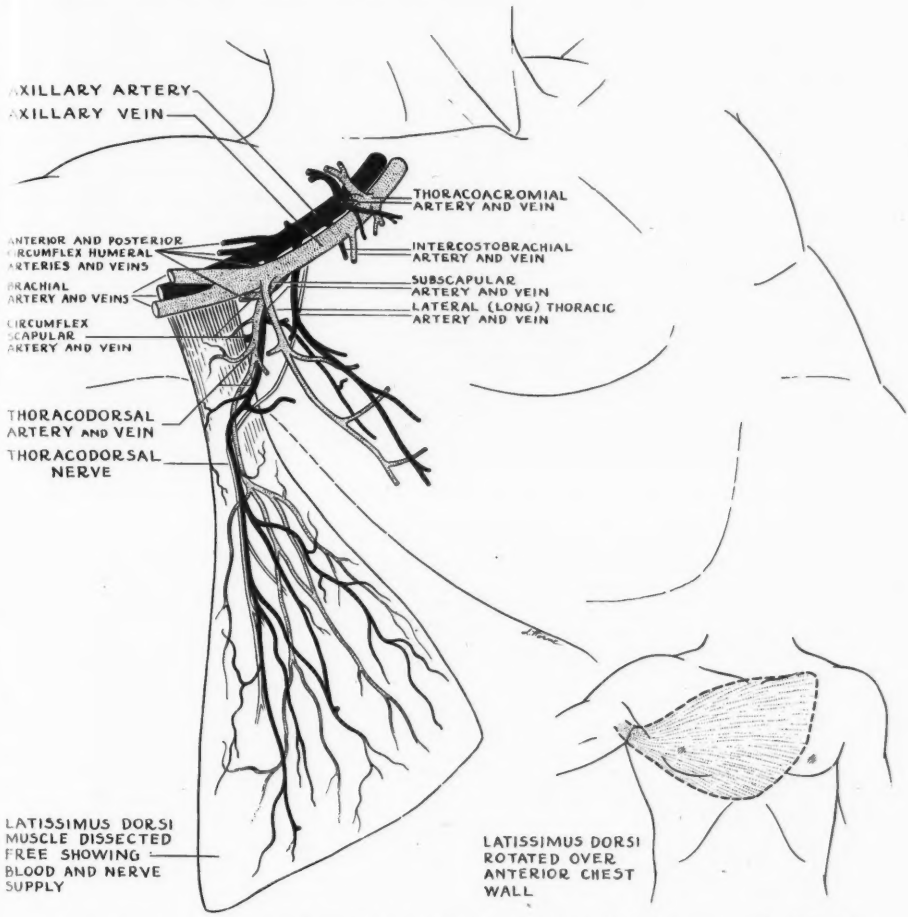
AXILLARY ARTERY
AXILLARY VEIN
THORACOACROMIAL ARTERY AND VEIN
ANTERIOR AND POSTERIOR CIRCUMFLEX HUMERAL ARTERIES AND VEINS
INTERCOSTOBRACHIAL ARTERY AND VEIN
BRACHIAL ARTERY AND VEINS
SUBSCAPULAR ARTERY AND VEIN
LATERAL (LONG) THORACIC ARTERY AND VEIN
CIRCUMFLEX SCAPULAR ARTERY AND VEIN
THORACODORSAL ARTERY AND VEIN
THORACODORSAL NERVE
LATISSIMUS DORSI MUSCLE DISSECTED FREE SHOWING BLOOD AND NERVE SUPPLY
LATISSIMUS DORSI ROTATED OVER ANTERIOR CHEST WALL

Fig. 1.—Drawing of the latissimus dorsi showing the intact blood and nerve supply following its mobilization.

Fascia Lata Graft.—Watson and James have pointed out the advantages of using a large square of fascia lata in the repair of a large thoracic wall defect (Fig. 3). It furnishes good support to the chest wall and aids in preventing pulmonary herniation. It does, however, require the use of a large pedicled flap of skin and subcutaneous tissue. For large defects this may be hazardous or impossible.

Use of the Latissimus Dorsi.—Various extracostal muscles have long been used in the repair of defects in the lateral and posterior aspects of the chest wall, but no effort has been made to extend their usefulness to include the ante-

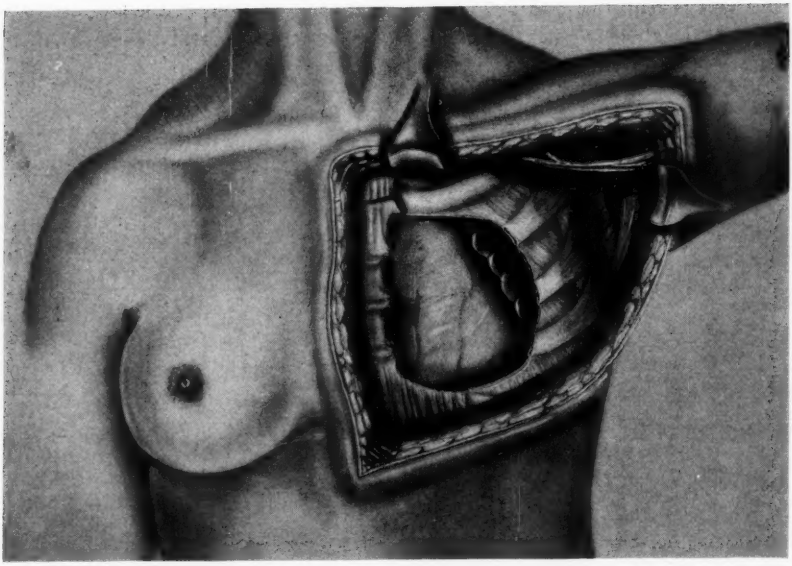

Fig. 2.—Defect of average size following removal of a three-rib segment of anterior chest wall.

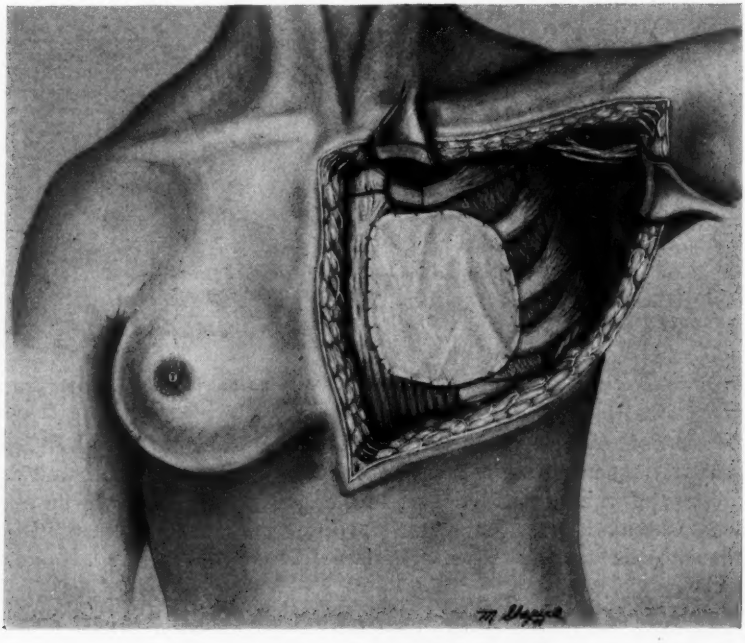

Fig. 3.—Application of the fascia lata graft.

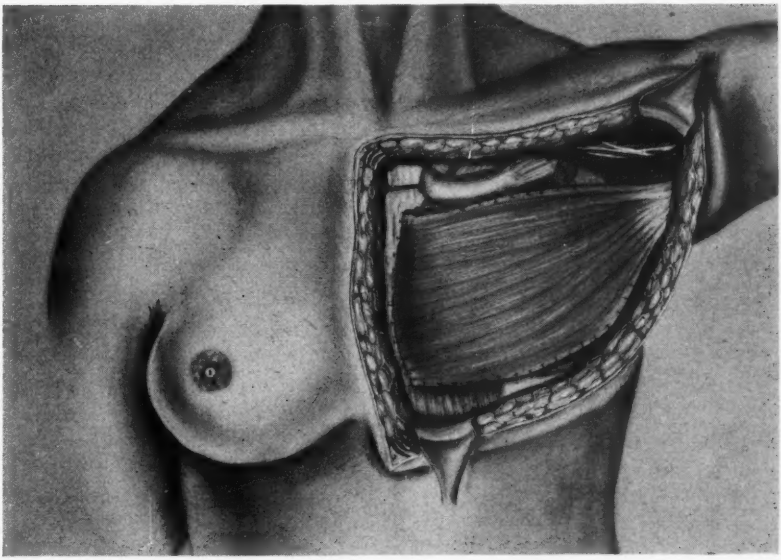

Fig. 4.—Transplantation of the latissimus dorsi to cover the defect.

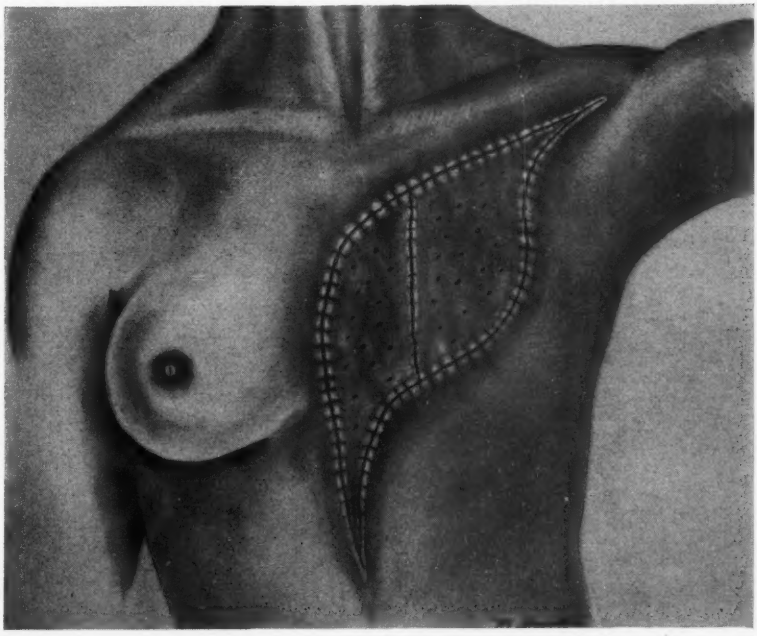

Fig. 5.—Completed procedure following application of split-thickness skin grafts.

rior aspect of the thorax. The latissimus dorsi, however, is capable of being transplanted to the anterior chest wall to assist in the repair of a defect at this site (Fig. 4). Exposure of the muscle is not difficult. If the excision of the neoplasm requires the dissection to extend to the anterior margin of the muscle, it can be mobilized by sharp dissection without additional skin incision. It is of some assistance if the patient is placed slightly on the opposite side prior to

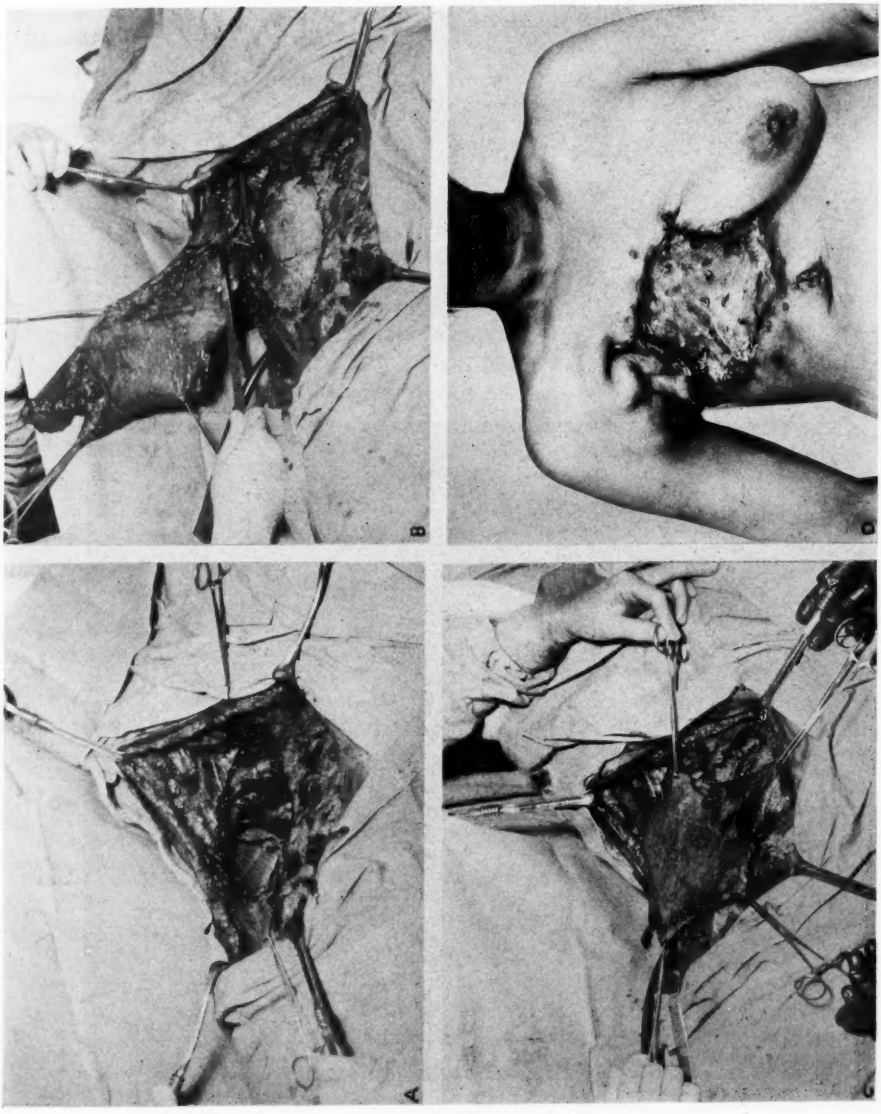

Fig. 6.—Photographs showing *A*, excision of lesion; *B*, fascia lata graft in place and latissimus dorsi mobilized (blood supply to muscle demonstrated by pointer); *C*, transplantation of muscle; *D*, twelfth postoperative day at first removal of dressing.

operation. Advantages of the use of this muscle are as follows: (a) It is readily accessible; (b) it can be completely mobilized, except for its insertion, and by means of sharp dissection, ligation of few perforating vessels, and incision of the lumbodorsal fascia which releases it from its origin, small blood loss is encountered; (c) it is capable of reaching any defect on the anterior aspect of the hemithorax; (d) it carries its own vascular and nerve supply during transplantation (Fig. 1); (e) it thus assures adequate nutrition, not only to itself, but to adjacent structures, such as the fascia lata graft beneath and split thickness skin grafts above, which require such assistance for survival; (f) there is little or no functional loss; (g) by furnishing another layer at the site of the defect, it aids in the support of the chest wall.

Some disadvantages may be associated with this procedure. It increases the magnitude of the operation by requiring additional dissection. This appears to be a minor consideration if the conditions for the operation are good. If the operation is being done for recurrent mammary cancer, the surgeon, who previously performed the radical mastectomy may have found it necessary to ligate and excise the subscapular vessels. If such is the case, the vascular supply would be almost entirely from the perforating branches of the intercostals, and mobilization of the muscle might leave its viability in question.

Split-Thickness Skin Grafts.—The application of split-thickness skin grafts needs little explanation (Fig. 5). The use of this method to produce an epithelial-covered area seems infinitely safer and easier than that of fashioning complicated pedicled flaps with the associated hazards so well known to most surgeons.

Customary pressure dressings are applied to the skin grafts, and serve also for temporary support to the chest wall for the first few postoperative days. Following their removal, no significant interference with cardiorespiratory function has been noted.

This procedure with various minor modifications has been carried out seven times with one postoperative death. This patient died suddenly sitting at her bedside on the third postoperative day. No autopsy was allowed.

Fig. 6.—Photographs showing *A*, excision of lesion; *B*, fascia lata graft in place and latissimus dorsi mobilized (blood supply to muscle demonstrated by pointer); *C*, transplantation of muscle; *D*, twelfth postoperative day at first removal of dressing.

SUMMARY

1. A method of repairing large defects in the anterior thoracic wall has been described. This method utilizes (a) a fascia lata graft, (b) complete mobilization and transplantation of the latissimus dorsi, and (c) split-thickness skin grafts.

2. Some advantages and disadvantages of this method are discussed.

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BLOOD AND NERVE SUPPLY TO THE ESOPHAGUS

An Experimental Study

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THE experimental study of the blood supply to the dog's esophagus was prompted by statements in the literature on esophageal surgery that the circulation of the esophagus is poor.[1-5] A recent study of end-to-end anastomosis of the esophagus under tension[6] demonstrated that considerable portions of the thoracic esophagus could be resected with extensive freeing of each segment, and successful end-to-end anastomosis performed. The present experiments attempted to define safe limits of mobilization of the esophagus in an effort to extend the scope of esophageal surgery, particularly in children. The character of the experiments also made it possible to study the effects of esophageal denervation.

The experiments consisted of mobilizing varying portions of the esophagus and evaluating the effect of this on (1) circulation, and (2) function. No comparable study has ever been made, to our knowledge. The nearest approach is the work of de Francesco,[7] who concluded that surgical denervation of the abdominal portion of the esophagus produced no significant alterations in tone or peristalsis. Although surgical opinion generally considers extensive mobilization of the esophagus dangerous,[1-5] Miller and Andrus[8] and Ochsner and Owens[9] mentioned freeing the thoracic esophagus well up into the mediastinum without serious consequences. Fischer[10] quoted Zaaijer as saying that it is possible to isolate the esophagus to a great extent without grave disturbance of its circulation.

The arteries supplying the esophagus of the dog have been described in detail by Saint[5] and Demel.[11] The upper cervical portion is nourished by branches of the thyroid artery. The lower cervical and upper thoracic divisions are supplied by branches from the subclavian artery. The mid-thoracic part of the esophagus receives its blood supply from the bronchoesophageal system, which arises as small branches from the aorta at the junction of its arch and descending portion. The lower thoracic esophagus is nourished by ascending and descending branches stemming from the aorta between the third and fifth intercostal arteries, while the remainder is supplied by diaphragmatic vessels and branches of the left gastric artery. The intrinsic esophageal arteries run longitudinally, and through these the extrinsic vessels of each group intercommunicate freely.

The motor innervation has two components, the pharyngeal branches of the glossopharyngeal nerve and the esophageal branches of the vagus nerve.[12]

The structure of the esophagus in the dog and the human being is roughly comparable, although in the dog striated muscle is more plentiful, comprising the upper third of the circular coat and the entire longitudinal layer. In ad-

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dition, the submucosa in dogs contains more mucous glands and fewer elastic fibers. These differences are minor, however, and the esophageal tissue of both man and dog is sufficiently similar in texture and consistency for surgical comparison.

For the sake of clarity, definitions of some of the terms used to describe the function of the esophageal muscle are given. Tonus is the resistance which the smooth muscle of the esophagus offers to distention.[12] Primary peristalsis is a progressive wave of contraction, carrying a bolus from the pharynx to the stomach. Secondary peristalsis, or segmentation, is a rhythmic nonprogressing contraction, not propelling food, and unrelated to tonus.

EXPERIMENTS

Operations were performed on sixty-three adult mongrel dogs of both sexes, ranging in weight from 12 to 30 pounds. In all experiments sodium pentobarbital was used for anesthesia, and pulmonary ventilation was maintained with intermittent positive pressure air insufflation given through an endotracheal tube. A right cervical and several different transpleural approaches were employed, depending on how large a portion of the esophagus was to be separated from its bed. In all instances the main trunks of the vagus nerves were visualized and dissected free from the esophagus. The actual mobilization of the esophagus was accomplished by sharp and blunt dissection. All possible delicacy was employed in handling the esophagus with instruments. Blood vessels, except for the azygos vein, were ligated only if they bled copiously.

The experimental series was divided into six groups, shown diagrammatically in Fig. 1.

Group I had an extensive procedure through a combined thoracocervical approach which permitted division of the esophageal blood and nerve supply between the cricoid cartilage and the diaphragm. *Group II* underwent a similar operation, except that the 2.0 cm. portion distal to the cricoid cartilage and another 2.0 cm. portion proximal to the diaphragm were left undisturbed. In *Group III* devascularization and denervation were limited to the thoracic esophagus from the diaphragm to a point about 2.0 cm. above the aortic arch. *Group IV* had the thoracic esophagus mobilized from the aortic arch, through the diaphragmatic hiatus, to the cardia. In this procedure the vessels extending 3.0 cm. along the greater and lesser curvatures were also divided and ligated. Both vagi were severed at the level of the diaphragm. In *Group V* the esophagus was mobilized between its mid-cervical and mid-thoracic points. In *Group VI* the upper 8.0 cm. of the cervical esophagus was freed from its bed.

Postoperative care included a milk diet for ten days, after which the animals received their usual ground horsemeat and dog biscuit. Follow-up consisted of fluoroscopic and roentgenologic studies two weeks after operation and at bimonthly intervals thereafter, until functional recovery occurred or death terminated the experiment. A thick mixture of barium sulfate, ground horsemeat, and milk, was used as a contrast medium.

Experimental results were evaluated clinically in terms of regurgitation of solids and liquids, and of survival. Objective studies consisted of fluoroscopy, roentgen films, gross and microscopic examination of specimens stained post mortem by the Loyez myelin, eosin-thionin, and eosin-cresyl violet techniques. The specimens were also studied by arterial injection with latex,* using a modification of Demel's method.[11] These were cleared in methyl salicylate and photographed by direct and reflected light.[13]

Our method of injecting the arterial system was as follows.

1. Anesthetize and exsanguinate dog.
2. Cannulate the abdominal aorta with a No. 13 needle below the renal arteries and secure it with three heavy ligatures.
3. Incise both external jugular veins and the inferior vena cava.

*Latex: S-765-A Red No. 1.

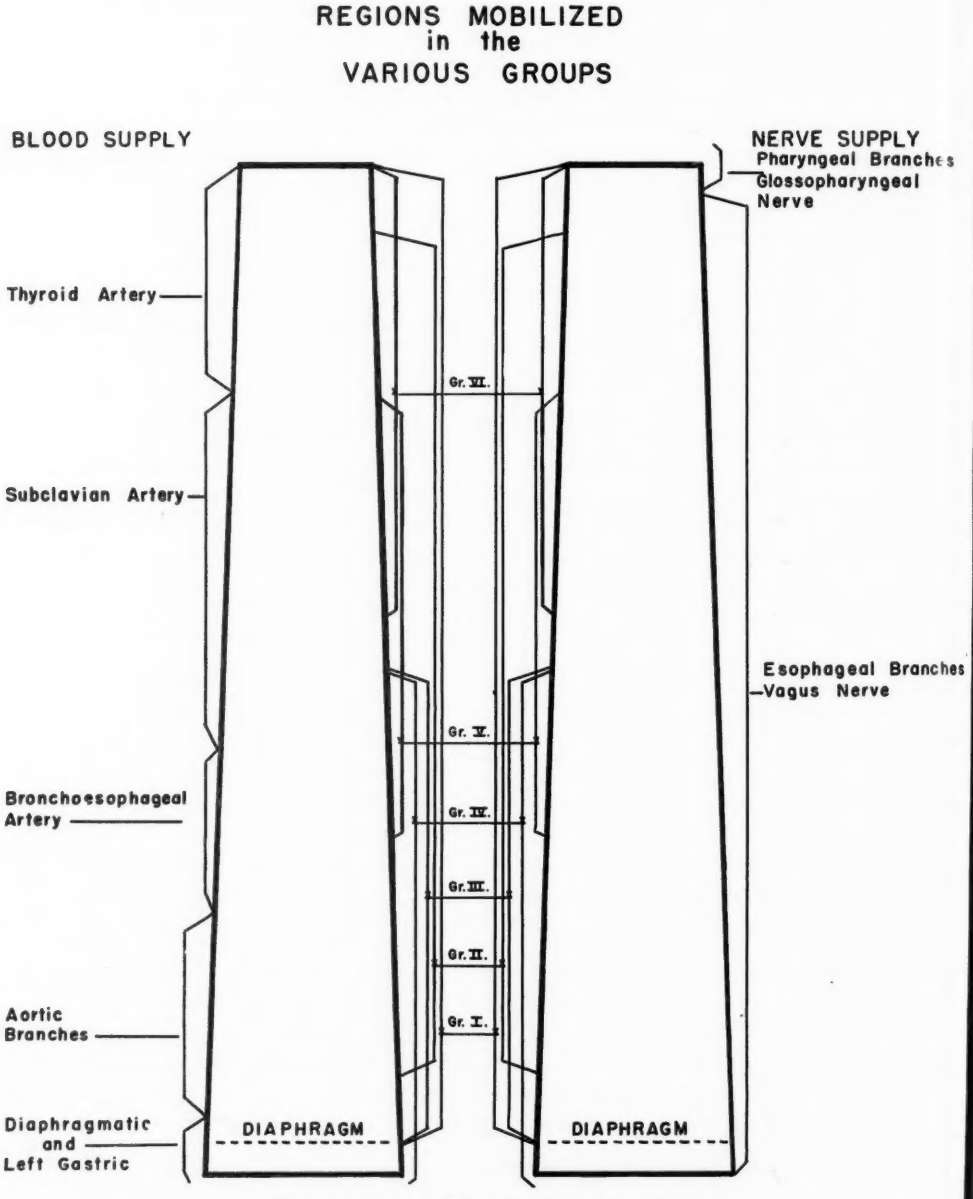

Fig. 1.—Regions mobilized in the various groups.

4. Flush the circulatory system with two to three gallons of cold isotonic saline solution while manipulating the neck, tongue, and stomach. Continue the flushing until flow from veins is clear and all visible veins contain no blood. The injection pressure should not exceed 250 mm. of mercury.

5. Mix latex* using either PC 520, a white, radiopaque material, or S-765-A red No. 1 for colored preparations; stir carefully in hot tap water and continue dilution until specific gravity does not exceed 1.005.

6. Inject under maximal pressure of 250 mm. of mercury for twenty to forty minutes.

7. Refrigerate entire cadaver for at least twelve hours.

8. Remove injected esophagus, fix in 10 per cent formalin, and clear through alcohol and benzene to methyl salicylate.†

EXPERIMENTAL RESULTS

The anatomic results of the esophageal mobilizations are given in Fig. 2. Complete observations on disturbed functions or pathologic findings are summarized as follows.

Group I.—In these eight dogs the mobilization cut off all the extrinsic esophageal blood supply except for the gastric and diaphragmatic branches and some pharyngeal connections (Fig. 1). Denervation included the pharyngeal branches of the glossopharyngeal nerve and the esophageal branches of the vagus.

Three dogs were sacrificed on the third postoperative day to obtain injected specimens. The remaining five died within eighteen days of operation. The average survival time was five days. One subject died of perforation of the esophagus, and the remaining four of pulmonary sepsis secondary to aspiration of saliva and ingested material.

Clinically, all subjects suffered from aspiration of overflowing esophageal contents secondary to cardiospasm and to lack of peristalsis. All began to regurgitate fluids and small samples of food on the first postoperative day and continued to do so until death.

Fluoroscopy of a typical subject two days after operation showed rapid ingestion of contrast medium, normal swallowing movements in the pharynx, but no primary peristaltic wave in the esophagus to grasp the bolus and carry it downward. The barium distended the entire esophagus until the structure filled and overflowed into the larynx. There was intermittent relaxation of the lower 2.0 cm. of the thoracic esophagus, permitting some barium to enter the stomach. In the roentgen films the esophagus resembled an elongated, inert sac with a rather uniform diameter of 3.0 cm. (Fig. 3).

Autopsies in this group revealed one mucosal ulceration, and one esophageal slough which encircled the esophagus and extended 3.0 cm. above and 7.0 cm. below the first rib. Microscopic examination of tissue near the slough showed acute inflammation with necrosis.

Summary.—Two dogs developed mucosal ulcerations and in one there was esophageal perforation. Aspiration and fatal pulmonary sepsis caused the death of four out of five unsacrificed dogs. In this group, neuromuscular impairment overshadowed vascular damage. Denervation caused cardiospasm as well as dilatation and loss of peristalsis in the esophageal segment mobilized.

Group II.—These twelve dogs had a mobilization of the esophagus which interrupted the blood supply from the subclavian, bronchoesophageal, and the upper aortic branches, and a denervation involving the vagus fibers from 2.0 cm. below the cricoid cartilage to 2.0 cm. above the diaphragm.

Fatal sequelae of devascularization were three perforations and three strictures of the esophagus. One dog died of pneumonia two days after operation. Another dog died of wound sepsis six days after operation. Four dogs lived to recover function. The stricture gradually disappeared in one subject.

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*Stock solutions sold by General Latex and Chemical Company, Cambridge, Mass.

†Time schedule for clearing: 70 per cent ethyl alcohol twenty-four to thirty-six hours; 95 per cent ethyl alcohol for another twenty-four to thirty-six hours; absolute alcohol, with two changes, twenty-four hours total; benzene U.S.P. twenty-four to forty-eight hours; store in Methyl salicylate, U.S.P., four parts, to benzyl benzoate, U.S.P., three parts, until clear.

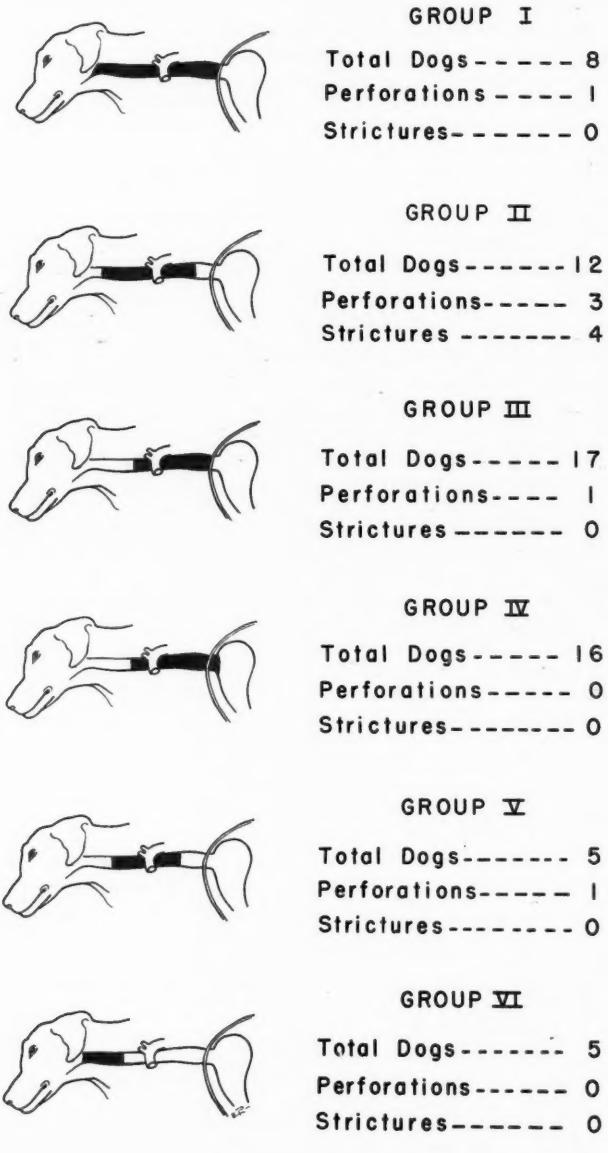


Fig. 2.—Anatomic changes following mobilization of various portions of the esophagus.

Regurgitation of solids occurred in eight animals, and in three fluids and saliva also were regurgitated. Three of these subjects developed strictures severe enough to cause death.

During fluoroscopy the contrast medium would be ingested and readily swallowed until the esophagus resembled an elongated, smooth-walled fusiform sac. Both the cervical and thoracic portions were involved, indicating lack of tonus.[14, 15] If more food were given it would push the esophageal contents through the cardiac sphincter into the stomach. If food were withheld, the dog would either regurgitate the esophageal contents, or else the latter would sluggishly enter the stomach during intermittent relaxation of the cardiac sphincter over a period of five to ten minutes.

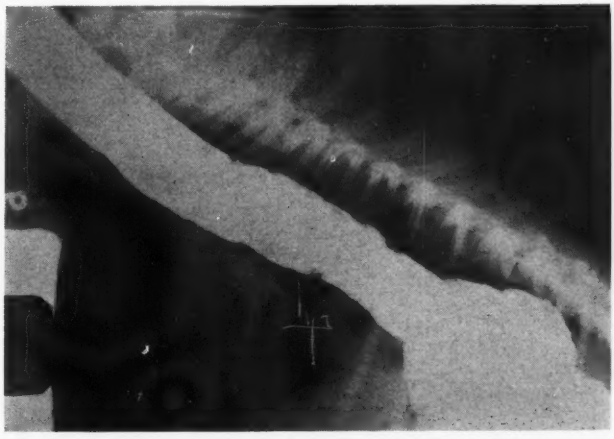


Fig. 3.—Lateral film two days after operation on dog in Group I. Note marked dilatation and absence of peristaltic waves.

The dilatation was temporary. The diameter of the cervical esophagus, normally 1.5 cm., increased to a maximum of 3.3 cm. at approximately six weeks after operation. During the same time the thoracic portion, normally 2.0 cm., attained a maximal diameter of 3.5 cm. (Fig. 4).

Effective peristalsis reappeared approximately ten weeks after operation, when it swept boli 5.0 to 6.0 cm. long down the esophagus to the stomach. Completely normal primary peristalsis was not present until about twenty-four weeks after operation, when boli were reduced to a normal 2.0 to 3.0 cm. and were carried without delay to the stomach.

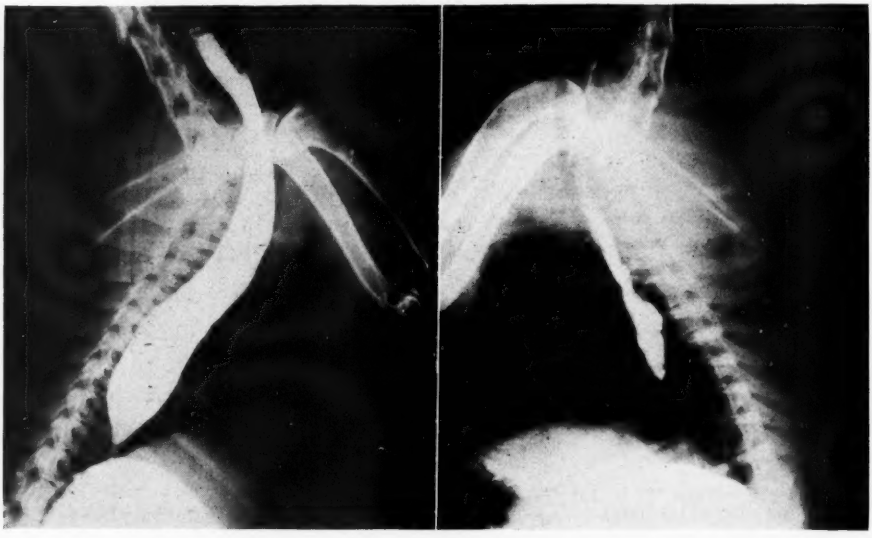
Minimal cardiospasm was seen in both films and fluoroscopy and disappeared after peristaltic recovery. It seemed more closely related to the absence of primary peristalsis than to true spasm, although it resembled Templeton's[16] description of cardiospasm.

Roentgenologic evidence of stricture at the level of the first rib appeared in four dogs within two weeks of operation. Each stricture consisted of a narrowed area 1.5 to 3.0 cm. long. One stricture disappeared gradually over a period of six months. In the other three dogs, stricture was severe enough to result in fatal complications.

Two animals, at necropsy six days after operation, were found to have esophageal necrosis with perforation. A third, sacrificed eight weeks after operation, showed a small, walled-off perforation and fibrous replacement of muscular and submucosal layers. Specimens from the three animals with strictures resulting in fatal complications exhibited an even more striking fibroblast proliferation into all layers, with degeneration of the epithelium to a one-layer cuboidal type. The strictures and perforations were found within 1.0 cm. of the first rib.

Four animals showing complete peristaltic recovery were sacrificed twenty-eight, thirty, thirty-four, and thirty-eight weeks after operation. All specimens were grossly normal in color and circumference, and were lined with healthy epithelium. Microscopic examination of sections taken at the levels of the first rib and the cardia showed completely normal structure.

The gross changes resulting from denervation were a marked dilatation and flabbiness of the esophagus. These were present in all dogs dying prior to complete peristaltic recovery. The esophageal circumference ranged from 3.0 to 4.0 cm. in the neck and from 4.0 to 5.0 cm. in the thorax, compared to a normal control specimen of similar length whose cervical and thoracic circumferences were 3.0 and 3.5 cm., respectively. This change was observed three days to eight weeks after operation. Normal-appearing ganglion cells of Auerbach's plexus were found in all specimens from dogs with complete functional recovery.



A. *B.*

Fig. 4.—*A*, Lateral film of dog in Group II five weeks after operation. Note dilatation, loss of peristalsis, and cardiospasm. *B*, Same dog, eleven weeks after operation. Note return of tonus and primary peristalsis.

Summary.—In these twelve animals there were three perforations and four strictures of the esophagus. One stricture disappeared and the dog completely recovered function. Denervation caused temporary cardiospasm as well as dilatation and loss of peristalsis in the mobilized esophageal segment. In the surviving six animals there was no incoordination between deglutition and peristalsis, and cardiospasm was minimal. Primary peristalsis returned in ten weeks and complete recovery occurred twenty-four weeks after operation.

Group III.—In this group of seventeen dogs, mobilization of the esophagus eliminated branches from the lower half of the subclavian artery, the bronchoesophageal arteries, and the aorta. Denervation included vagus fibers from 2.0 cm. above the aortic arch to the diaphragm (Fig. 1).

In this group, one animal died from improper anesthesia; two died in the first postoperative week, one from perforation of the esophagus and the second from wound infection. Two dogs were sacrificed in the first postoperative week to obtain injected specimens of esophagus. Nine died of pneumonia or distemper within twelve weeks of operation; none of these showed functional recovery. Three animals were followed for a longer period of time, and had complete recovery of esophageal function at twelve, twenty, and twenty-two weeks.

Solid food was regurgitated by four dogs and fluids by two, from the seventh to the eighteenth postoperative day, with one exception. This animal continued to regurgitate, with intermittent aspiration of food and saliva; death from pneumonia and multiple lung abscesses finally occurred three months after operation.

At fluoroscopy, the subjects had normal primary peristalsis to the level of the first rib. Below this point there was no peristalsis, and the esophagus dilated to a fusiform shape nearly three times its normal diameter. After the entire esophagus was fully distended, intermittent relaxation of the cardia occurred, sufficient to allow small boli to pass, but complete emptying did not take place. Gradual return of tonus began at about ten weeks, after the thoracic esophagus had reached a maximal diameter of 5.0 cm.

Beginning two weeks after operation, primary peristalsis and spasm of the lower 2.0 to 3.0 cm. of the esophagus were seen on all films. Primary peristalsis was too weak to be demonstrated fluoroscopically until approximately ten weeks after operation. Segmental contractions were minimal in number and strength two weeks after operation. These gradually increased in depth, but were too weak to be visualized by fluoroscopy until four to six weeks after denervation (Fig. 5).

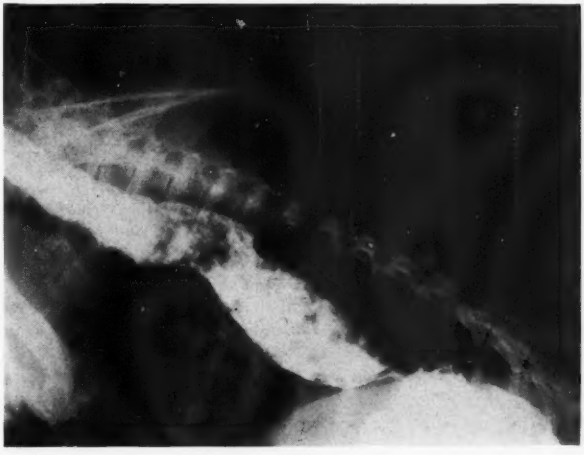


Fig. 5.—Lateral film of thoracic esophagus taken ten weeks after operation on dog in Group III. Note dilatation, absence of primary peristalsis, and cardiospasm.

Functional recovery occurred twelve weeks after operation in one dog. Another, who survived a similar length of time, had cardiospasm sufficiently severe to cause chronic aspiration. A third long-time survivor at sixteen weeks showed strong primary peristalsis, insufficient to overcome severe cardiospasm. At twenty weeks diminishing cardiospasm and dilatation were seen. At twenty-four weeks good function, with return of tonus and disappearance of cardiospasm, was demonstrated. At twenty-six weeks primary peristalsis transported boli 10.0 to 12.0 cm. long down the esophagus at normal speed without either residue in the upper thoracic esophagus or delay at the cardia.

At necropsy, severe circulatory insufficiency was manifested in one esophagus which perforated at the level of the first rib five days after operation. Ulcerations of the mucosa, up to 6.0 cm. in length, were observed in five specimens examined within two weeks of operation. Since they were found after the date at which perforation was usually seen, we considered them as phenomena of secondary importance.

Gross evidence of neuropathologic change was a striking flabbiness and dilatation of the thoracic esophagus. The circumference ranged from an average of 3.0 cm. three days after

operation to an average of 5.6 cm. three months after operation, compared to a circumference of 3.0 cm. in a control specimen of similar length. There was one death three months after operation from chronic aspiration caused by persistent cardiospasm.

Summary.—In a group of seventeen dogs, there was one anesthetic death, one death from perforation of the esophagus, and a third from pulmonary sepsis caused by aspiration secondary to cardiospasm. Denervation caused temporary cardiospasm as well as dilatation and loss of peristalsis in the mobilized esophageal segment. Primary peristalsis returned ten weeks after operation but persistent cardiospasm delayed complete functional recovery until twelve to twenty-six weeks after denervation.

Group IV.—In this group of sixteen dogs, esophageal mobilization extended from a point 2.0 cm. above the aortic arch, through the diaphragm, and along the greater and lesser curvatures of the stomach for 3.0 cm. Vessels divided included the lower esophageal branches of the subclavian artery, the bronchoesophageal, aortic, phrenic, and left gastric arteries, and the upper vasa brevia. Denervation compromised the entire vagal supply to the devascularized area (Fig. 1).

There was one anesthetic death. There were no deaths attributable to the devascularization and denervation in this group. Four animals were sacrificed to obtain injection specimens during the first week after operation. Four subjects died of distemper within three weeks of operation. One died of intussusception during the sixth postoperative week. Six dogs were studied twenty weeks or longer for functional recovery.

Four dogs began regurgitating food, and three of these saliva, two to four weeks after operation, and continued to do so for four to twelve weeks. However, no subject showed evidence of malnutrition or chronic aspiration.

All the dogs exhibited nearly uniform roentgenologic findings. Two days after operation peristalsis and segmentation were absent in the thoracic esophagus of two dogs. The contrast medium was ingested, swallowed, and carried by normal primary peristalsis to the thoracic esophagus, which would distend markedly until the dog either stopped eating or regurgitated. Eventually the esophagus emptied, usually after at least ten minutes. Spasm of the cardiac sphincter was more prolonged than in any of the other groups, and extremely active peristalsis in the upper esophagus was necessary to overcome this spasm. Although weak peristalsis was seen occasionally in all dogs fluoroscoped two to ten weeks after operation, moderately strong peristalsis was not present until twelve to fourteen weeks. At this time, cardiospasm was still too severe to allow prompt complete emptying of the esophagus.

Adequate functional recovery was characterized by active primary peristalsis, minimal dilatation of the lower eighth of the thoracic esophagus, and minimal cardiospasm. If food were given continuously, it would pass down the esophagus with a brief delay at the cardia. The terminal portion of the food bolus remained temporarily in the lower end of the thoracic esophagus. This phenomenon was observed in three long-term survivors twenty to twenty-nine weeks after operation. One dog was sacrificed during the twenty-eighth postoperative week because cardiospasm persisted and primary peristalsis had not reappeared in the thoracic esophagus. Two long-term survivors showed no discernible abnormalities thirty-two and thirty-four weeks after operation. Cardiospasm, gradually decreasing over a period of twenty-five to thirty-four weeks, appeared to be the disturbing factor in functional recovery.

Maximal dilatation of the thoracic esophagus measured in the roentgen films was 4.6 cm. and occurred usually thirteen weeks after operation. This coincided with the first fluoroscopic evidence of primary peristaltic return (Fig. 6). Tonus began to reappear at approximately ten weeks, and segmentation at five.

There were no deaths from insufficiency of the esophageal blood supply. Small mucosal ulcerations in the lower thoracic esophagus of three subjects, sacrificed at a date later than that at which perforations occurred in other groups, were, therefore, considered to be incidental findings.

Neuropathologic changes were suggested by the dilatation of the thoracic esophagus in seven of the twelve specimens examined two days to thirty-two weeks after denervation. The dilated esophagi ranged in circumference from 3.5 to 5.0 cm. The latter was obtained three weeks after operation.

Summary.—There were no strictures or perforations of the esophagus in this group of sixteen dogs. Denervation caused temporary cardiospasm and loss of peristalsis in the mobilized esophageal segment. Six of these dogs were studied to determine the time of functional recovery. This occurred in three dogs at twenty-six, and twenty-nine weeks, respectively. In the two which were followed for thirty-four weeks after denervation there was complete recovery, with no abnormalities demonstrable fluoroscopically. One dog showed no recovery of function and was sacrificed during the twenty-eighth postoperative week.

Group V.—In this group of five dogs, mobilization of the esophagus eliminated the ascending and descending branches of the subclavian artery. Denervation excluded vagus fibers from the mid-cervical to the mid-thoracic region (Fig. 1).

There was one death from esophageal perforation. Another death resulted from distemper eight weeks after operation. The three dogs that survived were studied for functional activity.

Fig. 6.—Lateral film of thoracic esophagus taken five weeks after operation on dog in Group IV. Note dilatation and absence of primary peristalsis.

None of the subjects showed evidence of food or fluid regurgitation during the immediate postoperative period.

At fluoroscopy the contrast medium was ingested and carried by primary peristalsis to the denervated portion, which dilated to nearly thrice its normal diameter. As long as feeding was continued, ingested boli pushed the contents of the inert, dilated esophagus into the stomach. At the end of feeding the distended area remained free of any peristalsis and emptied gradually over a period of several minutes. Peristalsis was absent approximately six weeks after operation. None of the dogs showed cardiospasm. Functional recovery with normal peristalsis appeared in the three long-term survivors eight to ten weeks after operation.

There was no gross evidence of neuropathologic change.

Summary.—There was only one instance of circulatory insufficiency, manifested by necrosis and perforation of the esophagus at the level of the first rib. Denervation caused dilatation and loss of peristalsis in the affected area. There was no cardiospasm. Functional recovery was observed eight to ten weeks after operation.

Group VI.—In this group of five dogs, mobilization of the upper cervical esophagus eliminated the esophageal branches of the thyroid artery. Thorough denervation affected efferent and afferent fibers of the glossopharyngeal and vagus nerves (Fig. 1).

There were no deaths from stricture, perforation, or aspiration. One dog died of distemper eight weeks after operation.

Clinically, these dogs showed no evidence of either aspiration or malnutrition. None regurgitated either saliva or ingested fluids.

During fluoroscopy the contrast medium was ingested, and with normal deglutition it entered the esophagus. It then dilated the inert upper esophagus until the contents sank to the unaffected portion, which emptied rapidly by primary peristalsis. All subjects showed temporary dilatation and loss of primary peristalsis in the denervated area, but no cardiospasm. Peristalsis returned and dilatation disappeared four to eight weeks after operation in three of the four long-term survivors. The fourth subject showed normal peristaltic activity at twelve weeks.

Summary.—There were no perforations, strictures, or other lesions indicating circulatory insufficiency in this group. Denervation caused local loss of primary peristalsis, but no cardiospasm. Complete recovery occurred, on the average, six weeks after operation.

DISCUSSION

Throughout the experiments we were impressed by the richness of the extensive longitudinal intrinsic arterial plexus demonstrated in the injected specimens (Fig. 7). We feel that these anastomosing intrinsic arteries were responsible for maintaining esophageal viability after localized portions of the esophagus had been denervated and devascularized.

In this series of sixty-three mobilizations of various portions of the esophagus in dogs we considered perforation and stricture to be evidence of serious vascular insufficiency. Mucosal ulceration, observed in several specimens in Groups III and IV subsequent to the expected time of perforation, presumably had little effect on either the viability or the function of the esophagus.

From our data (Figs. 1 and 2) we learned that either the cervical (that is, Group VI) or the thoracic esophagus from the cardia to a point about 2.0 cm. above the aortic arch (Groups III and IV) could be mobilized with safety. Devascularization of both the thoracic and cervical portions of the esophagus (that is, Groups I and II) caused serious vascular lesions in almost one-half of the animals. We found that no specific artery was vital to the esophageal circulation, nor was any one area more susceptible than another to necrosis after devascularization. Perforations were usually found in the midpoint of a devascularized segment.

Before considering the effects of denervation it would be well to summarize the swallowing mechanism. Normal deglutition consists of a series of coordinated reflexes mediated by extrinsic nerves from medullary and pontine centers. The *afferent* or sensory fibers of the reflexes are provided by the branches of the glossopharyngeal and trigeminal nerves, and by the pharyngeal and superior laryngeal branches of the vagus. These branches innervate the mucosa near the entrance to the pharynx. The *efferent* or motor fibers of the reflexes are supplied by the hypoglossal nerve to the tongue muscles, the pharyngeal branches of the glossopharyngeal nerve, and the pharyngeal and esophageal fibers of the vagus, to the muscles of the pharynx and esophagus, and the third division of the trigeminal nerve to the mylohyoid.

The dissection mobilizing the cervical esophagus from the thoracic inlet to the cricoid cartilage, including the posterior and lateral surfaces of the laryngeal portion, interrupted the afferent and efferent fibers in this segment. The interruption over so wide an area of the continuity of the reflex arcs responsible for the latter part of the pharyngeal and all of the esophageal stages of deglutition (that is, Groups I and VI) accounts for the absence of peristaltic

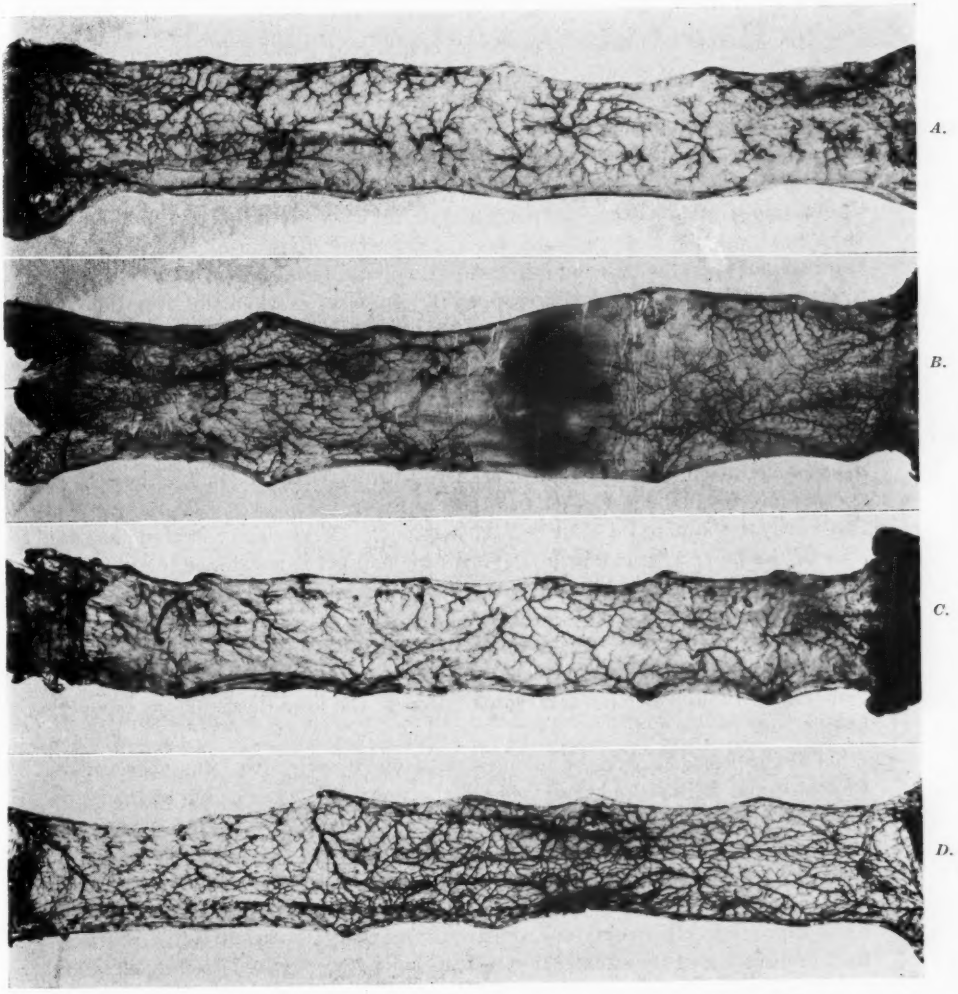


Fig. 7.—Representative specimens of esophagi with arterial blood supply injected. *A*, Normal control; note segmental arrangement of blood supply. *B*, Group I. Esophagus mobilized from cricoid cartilage to diaphragm. Note dilated longitudinal arteries extending into ecchymotic area. *C*, Group III. Esophagus mobilized from a point 2.0 cm. above the aortic arch to the diaphragm. Note dilated longitudinal arteries in the inferior half of the specimen. *D*, Group IV. Esophagus mobilized from a point 2.0 cm. above the aortic arch, through the diaphragm, to the cardia. Blood vessels of the greater and lesser curvatures ligated and divided, 3.0 cm. beyond the cardia.

response to swallowing in the denervated area. With normal primary peristalsis and no cardiospasm in the thoracic esophagus, and with thorough denervation of the lower pharyngeal and upper esophageal region, there was no aspiration (Group VI). With a similar, but more widespread denervation (Group I) there was cardiospasm and loss of peristalsis over the denervated area. We attribute the high incidence of fatal aspiration in Group I to cardiospasm and loss of peristalsis, and not to dissociation of pharyngeal and esophageal function.

The most fundamental concept of esophageal peristalsis is expressed in Starling's law: "Excitation of the gut at any point elicits contraction above and relaxation below the point of stimulation." Cannon[15] has postulated that this is a local phenomenon mediated by the myenteric plexus intrinsic to the esophagus. Transmission of primary peristalsis, except in the lower thoracic esophagus where smooth muscle predominated, is normally dependent upon the integrity of the vagus nerves. Simple division of the esophagus without vagal interruption does not stop peristalsis, while division of the esophageal vagus fibers results in complete temporary paralysis and dilatation of the upper two-thirds of the entire esophagus. In cases of vagal interruption the myenteric plexus assumes control after about seven days and recovery of peristalsis gradually follows.[14]

Although the extensive interruption of the esophageal fibers of the vagus in the various groups of dogs is adequate to explain temporary loss of peristalsis, it should be noted that all of the operations were accompanied by extensive devascularization, as well. This might well be expected to damage the cells of the myenteric plexus, or even the muscle fibers themselves. In this respect the work of Cannon and Burket[17] is pertinent. They demonstrated that the cells of the myenteric plexus of the intestine had great resistance to anoxemia, and could recover normal function even when absolutely deprived of their blood supply for periods up to seven hours. For this reason, and the fact that vagal interruption alone can account for peristaltic loss, we feel that partial removal of the esophageal blood supply was not the primary cause of this phenomenon, although it may have contributed to the long delay before recovery begins.

Cardiospasm, as well as changes in primary peristalsis, was observed by Cannon[14] and Milhiet[18] after division of the vagus below the origin of the recurrent laryngeal nerve. We noted, first, cardiospasm of varying severity in Groups I, II, III, and IV, but none in Groups V and VI. We also observed recovery from cardiospasm concomitant with the return of primary peristalsis in Group II at approximately fifteen weeks, but not in Groups III and IV until approximately twenty-one and twenty-six weeks, respectively. From our first finding we deduce that the cardiospasm noted resulted from a local and not a widespread disturbance of the esophageal innervation. From our second we learn that, although primary peristalsis reappeared almost simultaneously in Groups II, III, and IV, persistent cardiospasm greatly increased the average time required for functional recovery in the latter two groups.

It is evident from these experiments that a considerable portion of the esophagus can be separated from its regional blood supply with much less chance of serious complications than has been previously accepted. Surgical mobilization of one-half the entire esophagus is a safe procedure. Groups III and IV indicate that most of the thoracic esophagus can be freed even down to the stomach without compromising the blood supply. Group VI shows that the entire cervical esophagus can be freed without impairing its blood supply. To free the entire esophagus (cervical and thoracic) results in a very high incidence of vascular failure, as demonstrated in Groups I and II.

It should be emphasized that if a portion of the esophagus is to be resected, a proportionately larger segment of the esophagus may be freed from its vascular supply, since the area within the poorest circulation will be removed.[6]

It is clear from these experiments that after separating the esophagus from its bed, changes in function are observed which are caused by injury to the nerve supply. This division of nervous pathways for the most part results in a loss of peristalsis, which, fortunately, is only temporary. When the dissection is extended beyond the diaphragm there is prolonged cardiospasm unless the vagus nerves are carefully identified and preserved. However, even if the vagi are divided at this level, recovery from cardiospasm takes place after twenty-eight to thirty-four weeks. The occurrence of these functional changes is extremely important to remember in dealing with postoperative patients in whom a portion of esophagus has been freed.

The data of this experiment suggest solutions to several problems of esophageal surgery. A short benign stricture could be resected and repaired by end-to-end anastomosis. This has been reported by Gross,[19] Swenson.[20] Others have corrected atresia by mobilizing both segments of the esophagus extensively and performing an end-to-end anastomosis. Congenital short esophagus with diaphragmatic hernia has been successfully treated by mobilization of the esophagus upward to the aortic arch and downward through the diaphragm (as in Group IV), but leaving the vagus fibers intact, replacing the stomach in the peritoneal cavity, securing the esophagus in normal position with interrupted sutures, and repairing the diaphragmatic defect.[21] The satisfactory resection of esophageal malignancy and restoration of continuity with end-to-end anastomosis would depend on the presence of an early, small, well-localized lesion. Rienhoff[22] will soon report his experiences with this problem.

CONCLUSIONS

1. The esophageal blood supply is sufficiently rich to permit mobilization of large portions of the esophagus.

2. The cervical esophagus can be safely freed from its bed.

3. The thoracic esophagus can be safely mobilized from the aortic arch through the diaphragm without jeopardizing its blood supply.

4. When both the cervical and thoracic portions of the esophagus are mobilized, perforation or stricture occurs in nearly one-half of the animals.

5. The more extensively the esophageal fibers of the vagus are disturbed near the diaphragm, the more marked is the resultant cardiospasm.

6. Denervation of the esophagus causes loss of peristalsis followed by complete recovery from six to sixteen weeks after operation.

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BRONCHOSPIROMETRIC STUDIES IN BRONCHIECTASIS, BEFORE AND AFTER LOBECTOMY

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IN RECENT years the work of Cournand and Richards[1] and Pinner and associates[2] has changed the concept of the measurement of pulmonary function from that of a static measurement of vital capacity to the dynamic measurement of the many aspects of respiration including the various divisions of the lung volume, ventilation, and the diffusion and transport of the respiratory gases. The present study is concerned with the limited aspect of ventilation and oxygen uptake in a group of patients with bronchiectasis and the changes in these two functions following the surgical resection of diseased lung tissue.

MATERIAL AND METHODS

The over-all pulmonary ventilation was determined in 64 patients with bronchiectasis in whom the pulmonary disease had been demonstrated and delineated by Lipiodol bronchography of all the lobes. These patients ranged in age from 14 to 57 years. There were 33 male and 31 female patients. The results of the over-all function studies were compared with the over-all function obtained by the same method on 63 normal individuals within the same age range. In 44 of these patients lobectomy was carried out, and the over-all function was measured again postoperatively, usually one to three weeks after operation, and, in the majority of cases, again several months or even years later. In 39 of the group with bronchiectasis, bronchospirometric studies were carried out; in 21 of these preoperative and postoperative studies were made several months after operation. Five other patients were studied similarly by external spirometry and bronchospirometry before and after operation. These underwent lobectomy for bronchial adenoma (3), bronchial carcinoma (1), and pulmonary cyst (1).

External spirometry measuring over-all pulmonary ventilation was carried out with the patient in the basal condition using a Roth-Benedict water spirometer. With the patient resting in the supine position a record of quiet respiration was obtained over a four to five minute period; the resulting spirogram was used for analysis of the oxygen absorbed per minute, the respiratory rate, the depth of respiration (tidal air), and the average minute volume of respiration (M.V.R.), that is, the respiratory rate times the tidal air. The soda lime canister was then removed from the spirometer to lessen the resistance[3] and the patient was asked to breathe as deeply and as fast as possible for thirty seconds. A short recovery period was always recorded. From this record the maximum breathing capacity (M.B.C.) was calculated in terms of liters per minute. This method of measuring voluntary M.B.C. with the patient in the supine position gives consistently lower values than those obtained by Cournand[3] with the subject in the erect position. The breathing reserve at rest was derived (M.B.C. – M.V.R.) and the per cent breathing reserve calculated according to the formula $\frac{(\text{M.B.C.} - \text{M.V.R.})}{\text{M.B.C.}} \times 100$.

A Gebauer twin spirometer was used in the bronchospirometric determinations. Although a Zavod catheter was used in a few of the early determinations, a Norris type catheter[4] was used in the majority of the cases. The patients were not in a basal condition for this procedure. Local pontocaine anesthesia and fluoroscopic guidance were used, and the catheter was put in place with its tip in the left main bronchus.

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The division of function between the two lungs was calculated from the bronchospirometric record with reference to two factors: the per cent of the total oxygen absorbed and the per cent of the total volume of air moved by each lung. By referring these relative functions back to the resting oxygen absorption and tidal volume as determined by external spirometry under basal conditions, the probable resting function of each lung was estimated.

THE EFFECT OF BRONCHIECTASIS ON CERTAIN ASPECTS OF PULMONARY FUNCTION

Bronchiectasis is a chronic disease of the bronchi characterized by a cough usually productive of large amounts of purulent sputum. Consideration of the pathologic anatomy leads to an understanding of the physiologic changes which may occur with this disease. The affected bronchi are abnormally dilated and filled with mucopurulent secretion. The lesion of the bronchial walls may range from moderate dilatation with mild infection to enormous dilatation with loss of cilia, elastic tissue, and smooth muscle, and at times replacement of the walls by fibrous tissue[5, 6] with severe infection. Some part of the lung parenchyma is also usually involved by pneumonitis, hemorrhage, atelectasis, or fibrosis and scarring. Both ventilation and diffusion of respiratory gases to and from the blood may be impaired. The movement of air to and from the alveoli may be limited by the loss of elastic and smooth muscle tissue or frankly blocked by the accumulated secretions. The passage of respiratory gases to and from the blood may be further impaired by involvement of the alveoli in the disease process. Functional studies give some indication of the extent to which the normal pulmonary physiology is altered by these pathologic changes.

In this group of 64 patients with bronchiectasis there was little objective evidence obtained from external spirometry tracings alone that the respiration at rest was other than normal. In the majority of the cases the respiratory rate was less than 20 per minute, or within normal limits; in 11 it ranged between 20 and 24, and in 1 it was recorded as 30. Three patients who had widespread bronchiectasis complained of dyspnea at rest despite an apparently "normal" resting ventilation as recorded by external spirometry. Arterial oxygen content determinations would have contributed to a better understanding of the state of the resting pulmonary function.

The maximum ventilatory ability as measured by the voluntary maximum breathing capacity (M.B.C.) was frequently below normal (Fig. 1). To a certain extent it might be stated that the more extensive the disease the lower the maximum breathing capacity. The limited depth of respiration during maximum effort was largely responsible for the lower maximum breathing capacity values (Fig. 2). The rate and depth of respiration during maximum effort depend not only upon the patency of the airways but also upon the muscular strength of the individual and his ability to sustain muscular effort, which in turn are related to his general condition, his nutritional status, and the presence or absence of infection. Occasionally a striking increase in the maximum breathing capacity followed vigorous treatment which reduced infection and the amount of mucopurulent secretions and improved the general condition of the patient. Such treatment included bed rest, abolition of smoking, postural and bronchoscopic drainage, chemo- and antibiotic therapy, supplementary protein in the diet, iron

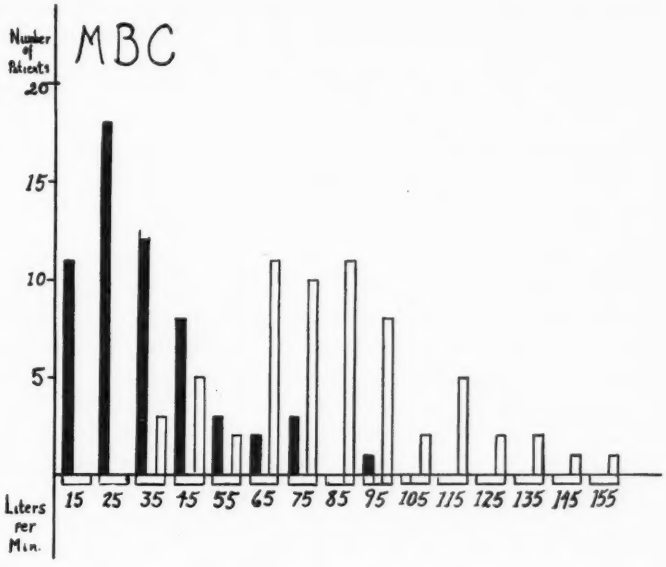

Fig. 1.—Maximum breathing capacities (black bars) obtained by maximum voluntary effort in 64 patients with bronchiectasis compared with maximum breathing capacities (white bars) obtained in 63 normal subjects in the same age group.

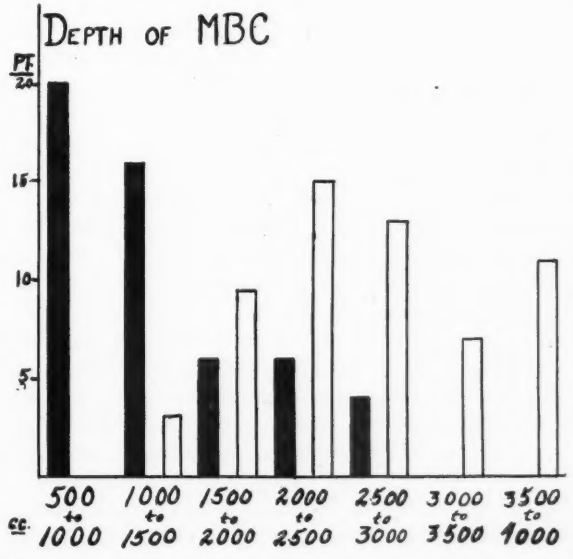

Fig. 2.—Depth of respiration (black bars) achieved during maximum voluntary effort by 64 patients with bronchiectasis compared with the depth of respiration (white bars) achieved by 63 normal subjects in the same age group.

administration, and blood transfusions. One patient with bilateral bronchiectasis had an initial maximum breathing capacity of 30 liters per minute; after one month of treatment this was increased to 66 liters per minute. A child with bilateral bronchiectasis doubled the depth of respiration on maximum effort after two weeks of intensive therapy. A fifteen-minute period of postural drainage alone was found to increase one patient's vital capacity by 400 c.c. (from 1,500 to 1,900 c.c.). On the other hand there was no change in the maximum breathing capacity of another patient after one month of similar intensive therapy.

External spirometry gives information only about the total lung function, and more specifically, only about total ventilatory function. Bronchospirometry, which separates the air moved by the right and left lungs simultaneously, provides the means of studying more closely the changes both in ventilation and in oxygen absorption occurring in pulmonary tissue diseased by bronchiectasis. It must be remembered that these studies will not necessarily give an exact picture of what is occurring in a diseased lung segment for it is not yet possible to measure the function of one segment alone. Bronchospirometry affords only a comparison of one lung with the other, a comparison of one lung containing diseased segments with the opposite lung which may be normal or partly diseased. At best it is not a question of comparing normal lung with abnormal lung but rather a problem of comparing normal lung with a combination of normal and abnormal lung.

Bronchospirometry was carried out on 39 patients with bronchiectasis. In 22 of these patients the bronchiectasis was essentially unilateral. In over one-half (12) of the cases of unilateral bronchiectasis the oxygen absorption from the diseased lung was impaired to a greater extent than the ventilation of air by this lung, that is, the per cent of the total oxygen uptake was less than the per cent of the total air ventilated. Of the remainder of the cases (10) the right:left ratios of oxygen absorption and ventilation were within normal limits in 4; oxygen absorption and ventilation were equally impaired in 2; while in 4 others the ventilation was reduced more than the oxygen absorption. We are unable to tell from these measurements how much the normal portion of lung on the affected side compensated in volume or in oxygen uptake for the diseased portion. At the moment it can be said that the data appear to suggest that in unilateral bronchiectasis oxygen absorption usually tends to be impaired to a greater extent than ventilation. That is, in the diseased lung the movement of air is less noticeably affected than the gaseous exchange across the alveolar membrane. Consideration of the pathologic anatomy alone might lead one to expect this to be the case. These results substantiate the work of Kay, Meade, and Hughes,[7] who likewise found that in bronchiectasis the uptake of oxygen from the diseased lung was generally proportionally less than the ventilation through that lung.

A comparison was made in the 22 cases of unilateral bronchiectasis between the function of the diseased lung and the function which might be expected if the diseased portion were completely functionless. A preliminary anatomic study was carried out on two pairs of lungs and an estimate was made of the

per cent division of the total lung volume by weight among the various lobes and their subdivisions. From these values the theoretical function which might be expected to remain in lung segments remaining on the operated side after lobectomy was calculated. This assumes, of course, that function parallels anatomic size, which is not necessarily true, particularly in an abnormal lung.

Two-thirds of the cases showed that the diseased lung ventilated a greater volume of air than might be anticipated on the basis of complete lack of function of the bronchiectatic areas. One-half of the total number showed a greater oxygen absorption from the diseased lung than predicted. From this it would appear that either the diseased segments were not completely functionless or that the undiseased portions had compensated for the loss of function to some extent. Both situations probably occur.

In summary, the presence of bronchiectasis in one or two lobes does not usually impair the resting ventilation appreciably. It does decrease the maximum breathing capacity below normal levels, particularly impairing the depth of respiration achieved, in a manner roughly proportional to the extent of the disease. The function of oxygen absorption is frequently more impaired by bronchiectasis than the function of ventilation. The divisions of both functions between the two lungs may not differ from the normal if the disease process is limited to only a few bronchopulmonary segments. It must be remembered that bronchospirometry measures only the relative functioning of the two lungs. We are not able to say with certainty how much adjustment has taken place in the undiseased portions of lung although compensatory increases in ventilation and oxygen absorption probably occur.

CHANGES IN PULMONARY FUNCTION FOLLOWING REMOVAL OF DISEASED SEGMENTS OF LUNG TISSUE

Questions then arise as to what occurs when a diseased segment or lobe of a lung is removed. Can an improvement in the over-all pulmonary function be expected with the removal of infected and distorted lung tissue, and what may happen to the function of the lobes of lung remaining on the operative side?

Lindskog[8] in 1939 stated that the removal of one or two lobes of a lung caused no permanent reduction of pulmonary volume, using the vital capacity and its component parts as criteria. Birath[9] studied both lobectomy and pneumonectomy patients two to thirteen years after operation and reported that the total lung volume was less reduced than the corresponding lung parenchyma, suggesting that distention of remaining lung tissue occurred postoperatively. The total dead space was found to be increased, probably due to the increased inflation of the lung occurring with distention of the parenchyma. Kay, Meade, and Hughes[7] concluded from 26 bronchospirometric studies carried out following lobectomy that the function of the remaining pulmonary tissue on the operated side was dependent largely on the postoperative pleural complications and that the postoperative function on the operated side in a patient with an uneventful convalescence may be within normal limits.

TABLE I. DATA OBTAINED PRE- AND POSTOPERATIVELY BY EXTERNAL SPIROMETRY ON FORTY-NINE PATIENTS WHO UNDERWENT PULMONARY RESECTION

RESECTED SEGMENTS	TIDAL VOLUME			MINUTE VOLUME			MAXIMUM BREATHING CAPACITY			DEPTH REACHED IN M.B.C.			COMPLICATIONS
	PREOP. (C.C.)	2 WEEKS POSTOP. (C.C).	LATER POSTOP. (C.C.)	PREOP. (LITERS)	2 WEEKS POSTOP. (LITERS)	LATER POSTOP. (LITERS)	PREOP. (LITERS)	2 WEEKS POSTOP. (LITERS)	LATER POSTOP. (LITERS)	PREOP. (C.C.)	2 WEEKS POSTOP. (C.C.)	LATER POSTOP. (C.C.)	
1. RLL partial	900	700	1250	8.1	8.4	12.5	70.0	57.0	101.0	2500	1350	2200	None
2. LUL	520	500	475	6.8	9.5	6.7	78.3	59.5	75.0	1400	850	1500	None
3. LLL	700	600		7.0	9.6		37.4	22.8		1700	950		
4. LLL	550		500	8.3		9.5	14.4		19.2	1200		800	None
6. LLL and lingula	550	450	850	11.0	9.5	9.4	32.0	17.0	35.2	1000	500	1100	None
7. LUL and LLL partial	500		400	6.0		5.6	16.2		19.2	900		800	TB
8. RLL, RML	475	600	620	7.1	7.2	7.5	44.0	21.1	31.5	1100	810	1050	Empyema
9. RLL, RML	700	560		7.7	10.6		48.0	28.8		1200	600		None
10. Lingula (bilateral disease)	800			16.0			69.0			1500			None
11. LLL (bilateral disease)	400	400	625	7.2	6.4	9.4	17.2	24.0	18.9	1075	1000	1350	Fever
12. RLL partial	650		600	13.0		10.2	41.8		63.7	1900		2450	Fever (9 days)
13. Lingula	880		1100	12.3		7.7	23.5		58.0	1175		2900	None
14. Lingula	900	700	600	15.3	16.8	7.2	44.0	40.0	39.1	1100	950	1150	None
15. RML	500		630	9.0		11.3	78.2		56.0	2300		2000	Fever (8 days)
16. LLL partial	300		550	5.4		8.8	36.4		30.8	1300		1100	None
17. LLL partial	750	500	660	9.7	9.5	5.9	52.0	33.6	52.8	2000	1200	2400	Atelectasis (5 days)
18. LLL	400	275	500	5.6	4.7	6.5	22.0	11.7	26.0	1000	650	1000	Fever (6 days)
19. LLL	700		700	6.3		7.0	20.6		30.0	1030		1500	Fever (11 days) atelectasis
20. LLL	650	500	460	11.1	12.0	11.1	48.4	26.4	45.6	1500	1200	1200	Empyema
21. LLL	750	1000	590	6.0	9.0	8.3	28.0	24.2	40.6	2000	1100	1450	Fever (11 days)
22. LLL	375	480	450	6.0	8.2	6.3	20.8	14.0	23.0	650	500	500	Empyema
22. Lingula	450	450	300	6.3	7.6	5.4	23.0	19.0	18.9	500	500	450	
23. RUL	1050	1250	1500	7.5	10.0	12.0	84.0	41.6	62.0	3500	1600	2200	None

24. LLL, lingula	540	700	600	3.8	7.0	4.8	37.8	28.8	64.5	1350	800	1900	Fever atelectasis (17 days)
25. LLL, lingula	650	600	425	7.8	6.6	5.5	25.5	33.6	27.3	1500	1050	1050	Fever (11 days)
26. LLL, lingula	440	260	400	9.7	9.4	6.9	27.9	11.2	20.8	775			None
27. LLL, lingula	350	220	580	8.1	7.3	8.1	21.0	12.2	18.5	700	435	1025	
28. RML, RLL	950	800	750	7.6	9.6	9.0	63.8	69.0	84.0	2900	1500	2800	Atelectasis (11 days)
29. RML, RLL	300	350	400	6.0	6.7	8.0	28.5	28.5	37.5	750	750	750	Atelectasis (8 days)
30. LLL, LUL, partial	1000	750	650	8.0	11.2	7.8	73.5	45.8	76.8	2300	1350	2400	Fever (13 days)
31. LLL (bilateral disease)		400	490		7.2	7.8		18.0	21.6		500	600	Atelectasis (7 days)
32. RLL partial (bilateral disease)	600	400	650	7.2	7.2	14.3	56.0	35.2	55.0	1750	800	1100	Fever (9 days)
33. RUL partial (bilateral disease)	600	575	450	8.4	6.9	5.4	17.0	16.0	11.6	850	800	580	
34. LLL partial, lingula	650		430	13.7		7.3	19.5		14.3			700	None
34. RLL, RML	490	460	650	6.9	10.6	9.0	14.7	18.4	20.4	560	575	850	None
35. Following LLL (bilateral disease), RML, RLL	640	500	750	6.4	6.5	9.0	37.5	25.2	21.6	1250	900	600	None
36. LLL	360	400	400	7.9	10.8	8.0	32.5	38.4	53.2	650	800	900	None
36. Lingula	400	300	600	8.0	9.6	12.0	53.2	52.2	51.6	900	900	1175	None
37. LLL partial	500		600	6.5		4.2	33.6		30.0	1400		1500	Fever (4 weeks)
38. LLL	350		460	4.5		9.2	32.2		58.1	700		1650	None
39. LLL	380		500	10.6		12.0	14.4		19.2	800		1200	None
40. LLL, lingula	500		500	8.0		7.0	23.8		42.0	625		1000	None
41. LLL, lingula	375	380	500	6.4	8.0	12.5	30.0	22.4	41.4	1500	800	1150	None
42. LLL partial	608		754	6.7		8.3	55.6		27.0				Empyema
43. LLL partial, lingula	550	400	450	9.4	8.8	5.9	36.0	19.2	54.4	1500	600	1600	Atelectasis (8 days)
44. LUL	550	650	600	7.7	10.4	6.6	33.0	28.8	42.0	1500	900	1500	None
45. RML, RLL partial	550	300		9.9	6.0		30.0	17.0		1000	425		Fever (11 days)
46. RLL	700	650	950	7.7	10.4	11.4	39.6	25.5	41.8	1650	750	950	
47. RLL (following LLL bilateral disease)	400	500	700	7.2	12.0	16.8	22.4	27.6	16.8	700	725	700	None
48. RML	540	400	500	7.0	5.2	6.0	20.0	21.0	39.6	2000	1050	1650	None
49. RUL	650		500	9.1		9.5	32.3		30.6	950		900	None

In our series preoperative and postoperative studies on 49 patients were compared for changes in over-all pulmonary function following resection of portions of diseased lung tissue (no pneumonectomies were included in this study) (Table I). The patients ranged in age from 14 to 57 years at the time of operation (average, 32.7 years). Operation was carried out in 44 patients for the treatment of bronchiectasis, in 3 for bronchial adenoma, in 1 for bronchiogenic carcinoma, and in 1 for the removal of a pulmonary cyst which occupied the lingula of the left upper lobe.

In the immediate postoperative period (two weeks or less), while the patient was still in the hospital, changes were noted both in the resting function and in the function with maximum voluntary effort. The resting respiration was usually more rapid than it had been prior to operation. There was little change in the depth of respiration at rest. There was, however, a marked decrease in the depth of respiration attained during maximum effort as well as in the vital capacity. Pain with deep respiration together with a lack of movement of the diaphragm on the operated side due to crushing of the phrenic nerve at operation were undoubtedly factors which contributed to these decreases. When complete healing had taken place and the thoracic contents had become stabilized, these difficulties were obviated and the over-all ventilatory function returned to its preoperative level or increased, as will be shown.

Comparison of the postoperative studies on 44 patients studied five months or more postoperatively with the preoperative tests showed that in this later period the resting respiration had returned to its preoperative character. The oxygen absorption at rest, the rate and depth of respiration, and, hence, the minute volume were the same as they had been preoperatively. Somewhat surprising results were encountered in the comparison of the maximum breathing capacities. In more than one-half the cases there had been no decrease in the depth of respiration attained during maximum voluntary effort; in 8 cases there had been a significant increase in the depth (more than 300 c.c.). The maximum breathing capacity is the product of the rate and the depth. Constancy or the increase of the depth coupled with either no change or an increase in rate during maximum effort resulted in the following facts: not only had the maximum breathing capacity returned to the preoperative values in 15 cases, but in 19 cases it had actually increased significantly (more than 15 per cent), the increase amounting to 147 per cent in 1 instance. In only 9 cases was there a significant decrease in maximum breathing capacity. These decreases were due primarily to a decreased depth of respiration attained during the test period. Two of the patients with decreased maximum breathing capacity had had empyema during the postoperative period; one had fever for eight days; 2 others had extensive bronchiectasis on the opposite side. There was no apparent reason for the decrease in the maximum breathing capacity in the remaining 5 cases.

The fact that the resting respiration (ventilation and oxygen uptake) was the same as it had been preoperatively in all cases and that the maximum breathing capacities were the same or better in 82 per cent of the cases suggests that compensatory changes may have taken place in the remaining lung tissue. Bronchospirometric studies furnish a means of studying these changes more

closely. There are three possibilities with respect to the function of the remaining lung tissue: (1) No change occurs in the ratios of the air moved or the oxygen absorbed by the two lungs, in which case any compensatory changes or increases in volume or in oxygen uptake are contributed to equally by both the operated and unoperated sides; (2) the per cent of the total ventilation and oxygen absorption of the operated side is reduced with the result that compensatory changes occur largely in the unoperated lung; (3) there is an increase in the per cent of the total function contributed by the remaining segment of lung on the operated side, which means that it is this segment which is providing a large part of the compensation.

A comparison of the percentage distribution of the movement of air and the oxygen uptake as determined by bronchospirometry in 24 cases pre- and postoperatively showed that all three of the aforementioned possibilities occurred, but with different frequencies (Table II). Moreover, the changes in ventilation and in oxygen absorption did not parallel each other in every case.

In 10 cases the per cent of the total volume of air moved by the operated lung was significantly decreased postoperatively, which can only mean that any over-all increases in the depth of ventilation which occurred were provided by the unoperated lung. In 10 cases there was no change in the ratios of the volume of air ventilated by each lung. This means that either the resected lobe had been completely without function prior to operation or that the remaining lung segments on the operated side assumed the function formerly contributed by the diseased lung tissue. This does not mean that the ventilatory function of the operated lung had become normal, but merely that the division of function between the two lungs was the same as it had been preoperatively. Very infrequently (in 4 cases) there was an actual increase in the proportion of air moved by the operated lung.

The findings concerning the ratio of the oxygen absorbed by the two lungs were somewhat different and did not necessarily parallel the changes in ventilation. In 12 cases there was a decrease in the per cent of oxygen absorbed by the operated side, in 6 cases there was an increase, and in 6 cases there was no change.

Of the 24 patients on whom bronchospirometry was done before and after resection of diseased lung tissue, 18 had some reduction either in the per cent of the oxygen uptake or per cent of total ventilation contributed by the operated lung. Four of the 18 had a reduction in both functions. Six of the 24 patients had either no change or an increase in the functions of ventilation and oxygen absorption of the operated lung. The findings in the 14 patients who had had partial lobectomies or a lobectomy plus resection of a segment of a second lobe did not differ from those of the entire group.

Certain other facts might also be pointed out. After the surgical removal of the diseased lung tissue, the ratios of the air moved by each lung and the oxygen absorbed did not usually return to normal unless they were normal preoperatively. In 4 cases only was the resulting ratio of the ventilation by the right and left lung normal (right 53 per cent ± 5: left 47 per cent ± 5); in 3 of these the ratio had been normal preoperatively and had not been changed

TABLE II. DATA OBTAINED PRE- AND POSTOPERATIVELY BY BRONCHOSPIROMETRY ON THIRTY-FIVE PATIENTS WHO UNDERWENT PULMONARY RESECTION

RESECTED LUNG SEGMENTS	OXYGEN CONSUMPTION						TIDAL VOLUME						POSTOPERATIVE COMPLICATIONS
	PREOPERATIVE			POSTOPERATIVE			PREOPERATIVE			POSTOPERATIVE			
	RIGHT-LEFT		RATIO	RIGHT-LEFT		RATIO	RIGHT-LEFT		RATIO	RIGHT-LEFT		RATIO	
1. RLL partial	216	184	54:46				540	360	60:40				
2. LUL	149	67	69:31				260	260	50:50				
3. LLL	184	104	64:36				525	175	75:25				
4. LLL	137	113	55:45				386	164	61:39				
6. LLL, lingula	108	75	59:41				372	248	60:40				
7. LUL, part LLL	248	65	79:21				380	120	76:24				
8. RML, RLL	103	87	54:46				252	223	53:47				
9. RML, RLL	92	138	40:60				231	469	33:67				
10. Lingula (bilateral disease)	88	78	53:47				272	188	59:41				
11. LLL ((bilateral disease)	144	62	70:30				164	136	41:59				
12. RLL partial	87	66	57:43	158	68	70:30	351	299	54:46	312	288	52:48	Fever (9 days)
13. Lingula	154	52	71:29	149	104	59:41	625	255	71:29	682	418	62:38	None
14. Lingula	116	84	58:42	114	102	53:47	513	387	57:43	390	210	65:35	None
15. RML	155	345	31:69	126	294	30:70	276	199	58:42	353	277	56:44	Fever (8 days)
16. LLL, basal segments	110	110	50:50	143	40	78:22	150	150	50:50	429	121	78:22	None
17. LLL, basal segments	91	84	52:48	193	82	70:30	563	187	75:25	488	172	74:26	Atelectasis (5 days)
18. LLL	182	111	62:38	151	62	71:29	252	148	63:37	335	165	67:33	Fever (6 days)
19. LLL	138	39	78:22	175	28	86:14	406	294	58:42	406	294	58:42	Fever and atelectasis (11 days)
20. LLL	238	45	84:16	179	14	93: 7	520	130	80:20	382	78	83:17	Empyema
21. LLL	146	54	73:27	199	41	83:17	450	300	60:40	460	130	78:22	Fever (11 days)
22. LLL, lingula	114	69	62:38	127	26	83:17	195	180	52:48	373	77	83:17	Empyema
23. RUL	119	164	42:58	156	144	52:48	430	620	41:59	570	930	38:62	None
24. LLL, lingula	227	93	71:29	219	81	73:27	350	190	65:35	462	138	77:23	Fever and atelectasis (17 days)
25. LLL, lingula	120	80	60:40	144	139	76:24	416	234	64:36	267	383	56:44	Fever (11 days)
26. LLL, lingula	113	30	79:21	124	26	83:17	246	194	56:44	273	52	84:16	None
27. LLL, lingula	138	75	65:35	140	93	60:40	178	172	51:49	435	145	75:25	
28. RML, RLL	78	182	30:70	107	136	49:51	360	590	38:62	420	330	56:44	Atelectasis (11 days)
29. RML, RLL	92	133	41:59	63	127	33:67	138	162	46:54	168	232	42:58	Atelectasis (8 days)
30. LLL, LUL, except for apical segment	158	85	65:35	222	94	70:30	580	420	58:42	428	222	66:34	Fever (13 days)
31. LLL (bilateral disease)			57:43	138	62	69:31			42:58	358	132	73:27	Atelectasis (7 days)
32. RLL (bilateral disease, partial)	86	120	42:58	154	166	48:52	228	372	38:62	272	378	42:58	Fever (9 days)
33. RUL partial (bilateral disease)	22	78	22:78	69	169	29:71	220	280	44:56	180	270	40:60	None
34. Following LLL partial RLL, RML	190	60	76:24	175	75	70:30	288	142	67:33	253	397	39:61	None
35. Following LLL RML, RLL	79	114	41:59	156	117	57:43	282	358	44:56	413	337	55:45	None

by operation. In one case there had been an increase in the per cent of the air moved by the remaining segment of lung on the operated side. Kay and associates[7] found normal ratios postoperatively in some of their patients; the number was not stated.

An attempt was made to correlate the postoperative course of these patients with the changes in the functions of oxygen uptake and ventilation of the operated lung as shown by bronchospirometry. Of the 8 patients with an absolutely uneventful postoperative course (that is, with no fever after the third postoperative day and no x-ray evidence of atelectasis after the first four to five days postoperatively), 4 had some decrease in function. All 4 showed a reduction in the per cent of total air ventilated by the operated lung, but only 2 had a reduction in the per cent of total oxygen absorption.

Six patients had x-ray evidence of atelectasis for 5, 7, 8, 11, 11, and 17 days, respectively. Of these, 3 had a reduction in the per cent of total oxygen taken up by the operated lung with no change in ventilation. One had a decrease in both of these functions. Surprisingly, 2 patients in whom the atelectasis was present for 11 and 17 days had no impairment in oxygen absorption in the remaining segment after it had re-expanded.

Of the 7 patients who had fever alone for 7 to 21 days postoperatively, with no apparent etiology other than the chest lesion, 4 showed some reduction in function of the operated lung. All 4 showed a decrease in the per cent of oxygen taken up by the remaining lung segment and only 2 had a decrease in the per cent of air moved.

The 2 patients with postoperative empyema and bronchopleural fistulas did badly. Both had very little function remaining on the operated side either with regard to oxygen absorption or ventilation of air.

Considering these same bronchospirometric findings from another viewpoint, of the 12 patients who had reductions in per cent of total oxygen use of the operated lung following resection, 9 had some postoperative complication (atelectasis, fever, or empyema); of the 10 patients with a reduction in the per cent of total ventilation by the operated lung, only 6 had some postoperative complication, 4 having what was considered to be an uneventful postoperative course. However, not every patient with postoperative complications suffered impairment of oxygen absorption of ventilatory ability. To summarize, reductions in the ventilation may occur even with an uneventful postoperative course, whereas reductions in oxygen uptake are much more likely to be associated with complications such as fever, atelectasis, or empyema. It is also possible for a patient to have such complications and to suffer no impairment in the functions of ventilation or oxygen uptake of the operated lung.

SUMMARY AND CONCLUSIONS

Two aspects of respiratory function were studied, namely, ventilation and oxygen uptake, in a group of 64 patients with bronchiectasis and 5 patients with other pulmonary disease. These were compared with 63 normal subjects.

In bronchiectasis it was found that the maximum breathing capacity, as shown by external spirometry, was reduced below normal in a manner roughly

proportional to the extent of the disease. This reduction was due in the main to a decrease in the depth of respiration attained during maximum voluntary effort.

Bronchospirometric studies showed that in bronchiectasis the oxygen uptake in the diseased lung was often more reduced than the volume of air moved. The reductions in these functions were usually less than would be predicted from anatomic studies if the diseased lung were completely functionless, indicating that there was probably both some residual function in the diseased portions and some compensation on the part of the undiseased segments.

Although the reduction in the per cent of air moved on the diseased side was made up, in part, by an increase in the per cent of the total air moved by the undiseased lung, the over-all ventilation was normal only during quiet respiration. It was reduced during effort.

Lobectomy or surgical resection of diseased segments of lung usually did not change the over-all ventilation to any extent, as shown by external spirometry. Often, there were increases in the maximum breathing capacity after resection. Occasionally, there were decreases.

However, bronchospirometry showed that in the majority of cases (18 out of 24) there was some further loss of function on the diseased side after the surgical removal of the diseased segments of lung. These reductions in ventilatory function or in the per cent of the total oxygen absorbed were balanced by increases in these functions in the opposite undiseased lung.

In almost every case a reduction in the per cent of the oxygen absorbed on the operated side was associated with some postoperative complication such as empyema, atelectasis, or fever, attributable to no cause other than the chest lesion. Reductions in the per cent of the total air moved on the operated side occurred, on the other hand, following an apparently uneventful postoperative course. Nevertheless, it is possible for postoperative complications such as atelectasis and fever attributable to the chest lesion to occur and to have no change in the per cent of the oxygen absorbed or the per cent of the total air ventilated by the operated side.

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AN INSTRUMENT FOR PASSING LIGATURES

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AN INSTRUMENT has recently been devised for passing ligatures about deeply placed structures. This report is for the purpose of describing the instrument.

The over-all length of the ligature passer is 10 inches (Fig. 1). It is $\frac{5}{8}$ inch in breadth near the proximal (handle) end, and it tapers to a breadth of $\frac{3}{16}$ inch near the curved distal end. The curve near the distal end is one quarter

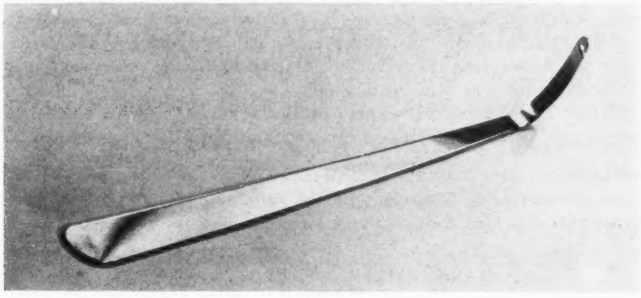

Fig. 1.

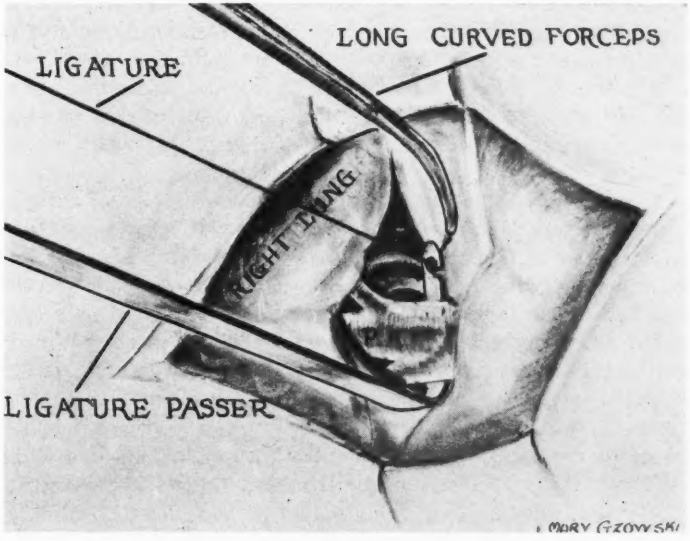

Fig. 2.

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of a circle 2 inches in diameter. The instrument is made of malleable stainless steel, and is $\frac{3}{32}$ inch in thickness. The ligature slot opens at a distance of $\frac{1}{4}$ inch from the rounded distal tip and is cut obliquely.

TECHNIQUE

The ligature passer has been used in ligation of the pulmonary artery and veins in pneumonectomy and pulmonary lobectomy at the Montreal General Hospital.

After the appropriate segment of artery or vein has been dissected free, the tip of the unthreaded instrument is passed under the vessel. The ligature is then placed in the slot, and upon withdrawing the instrument, is carried about the vessel. It has been found most convenient to introduce the ligature into the slot in the following way: As the surgeon passes the tip of the instrument about the vessel to be ligated, an assistant holds the ligature under tension by grasping one end in a long curved forceps and the other end in his fingers. Upon placing the ligature in the slot the assistant releases the free end and the surgeon simultaneously withdraws the instrument, passing the free end of the ligature about the vessel (Fig. 2). Due to its malleability the surgeon can easily alter the curve of the instrument as occasion demands.

It is felt that this ligature passer may be used to advantage in a number of situations (for example, thorax, pelvis) where the structures to be ligated are deeply placed.

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